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## SCIENTIFIC OPINION

# Scientific Opinion on Dietary Reference Values for carbohydrates and dietary fibre[1]

**EFSA Panel on Dietetic Products, Nutrition, and Allergies (NDA)**[2, 3]

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## ABSTRACT

This Opinion of the EFSA Panel on Dietetic Products, Nutrition, and Allergies (NDA) deals with the establishment of Dietary Reference Values for carbohydrates and dietary fibre. Nutritionally, two broad categories of carbohydrates can be differentiated: "glycaemic carbohydrates", i.e. carbohydrates digested and absorbed in the human small intestine, and „dietary fibre", non-digestible carbohydrates passing to the large intestine. In this Opinion, dietary fibre is defined as non-digestible carbohydrates plus lignin. The absolute dietary requirement for glycaemic carbohydrates is not precisely known but will depend on the amount of fat and protein ingested. The Panel proposes 45 to 60 E% as the reference Intake range for carbohydrates applicable to both adults and children older than one year of age. Although high frequency of intake of sugar-containing foods can increase the risk of dental caries, there are insufficient data to set an upper limit for (added) sugar intake. Based on the available evidence on bowel function, the Panel considers dietary fibre intakes of 25 g/day to be adequate for normal laxation in adults. A fibre intake of 2 g/MJ is considered adequate for normal laxation in children from the age of one year. Although there is some experimental evidence that a reduction of the dietary glycaemic index and glycaemic load may have favourable effects on some metabolic risk factors such as serum lipids, the evidence for a role in weight maintenance and prevention of diet-related diseases is inconclusive.

## KEY WORDS

Carbohydrates, dietary fibre, sugars, added sugars, glycaemic carbohydrates, oligosaccharides, starch, lignin, glycaemic index, glycaemic load, dietary requirements, blood lipids, lipid profile, glucose tolerance, insulin sensitivity, body weight, type 2 diabetes, blood pressure, cardiovascular disease, coronary heart disease, dental caries, gastrointestinal function, colorectal cancer, mineral absorption

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## SUMMARY

Following a request from the European Commission, the EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA) was asked to deliver a scientific opinion on Population Reference Intakes for the European population, including carbohydrates and dietary fibre.

Nutritionally, two broad categories of carbohydrates can be differentiated: "glycaemic carbohydrates", i.e. carbohydrates digested and absorbed in the human small intestine, and "dietary fibre", non-digestible carbohydrates passing to the large intestine.

The main glycaemic carbohydrates are monosaccharides, disaccharides, malto-oligosaccharides, and starch. In this Opinion the term "sugars" is used to cover monosaccharides and disaccharides. The term "added sugars" refers to sucrose, fructose, glucose, starch hydrolysates (glucose syrup, high-fructose syrup) and other isolated sugar preparations used as such or added during food preparation and manufacturing. Sugar alcohols (polyols) such as sorbitol, xylitol, mannitol, and lactitol, are usually not included in the term "sugars". However, they are partly metabolised and included in "carbohydrates" according to the European legislation.

In this Opinion, dietary fibre is defined as non-digestible carbohydrates plus lignin, including non-starch polysaccharides (NSP) – cellulose, hemicelluloses, pectins, hydrocolloids (i.e., gums, mucilages, β-glucans), resistant oligosaccharides – fructo-oligosaccharides (FOS), galacto-oligosaccharides (GOS), other resistant oligosaccharides, resistant starch – consisting of physically enclosed starch, some types of raw starch granules, retrograded amylose, chemically and/or physically modified starches, and lignin associated with the dietary fibre polysaccharides.

Main dietary sources of sugars are fruits, berries, fruit juices, some vegetables, milk and milk products, and foods containing added sucrose and starch hydrolysates (e.g., glucose syrup, high-fructose syrup) such as carbonated beverages and sweets. Main dietary sources of starch are bread and other cereal products, potatoes, tubers and pulses.

Data from dietary surveys show that average carbohydrate intakes in European countries in children and adolescents varied between 43 to 58 E%, and from 38 to 56 E% in adults. Average intakes of sugars varied between 16 to 36 E% in children and adults.

Whole grain cereals, pulses, fruit, vegetables and potatoes are the main sources of dietary fibre. Average dietary fibre intakes varied from 10 to 20 g per day in young children (<10 to 12 years), from 15 to 30 g per day in adolescents, and from 16 to 29 g per day in adults. Average intakes of dietary fibre per MJ ranged from 1.7 to 2.5 g per MJ in (young) children and from 1.8 to 2.9 g per MJ in adults.

### Total and glycemic carbohydrates

As energy balance is the ultimate goal, dietary reference values for carbohydrate intake cannot be made without considering other energy delivering macronutrients and will be given as percentage of total energy intake (E%). The absolute dietary requirement for glycaemic carbohydrates is not precisely known but will depend on the amount of fat and protein ingested. Generally, an intake of 50 to100 g per day will prevent ketosis. An intake of 130 g per day for both children (>1 year) and adults has been estimated to be sufficient to cover the needs of glucose for the brain. However, these levels of intake are not sufficient to meet energy needs in the context of acceptable intake levels of fat and protein.

Intervention studies provide evidence that high fat (>35 E%), low carbohydrate (<50 E%) diets are associated to adverse short- and long-term effects on body weight, although data are not sufficient to define a Lower Threshold of Intake (LTI) for carbohydrates. Similarly, high carbohydrate diets tend

to induce adverse effects on the blood lipid profile, but there is an insufficient scientific basis for setting a Tolerable Upper Intake Level (UL) for total carbohydrates. The Panel therefore comes to the conclusion that only a Reference Intake range can be given for total carbohydrate intake, partly based on practical considerations (e.g. current levels of intake, achievable dietary patterns).

Based on the above considerations the Panel proposes 45 to 60 E% as the Reference Intake range for carbohydrates. Diets with glycaemic carbohydrate contents of 45 to 60 E%, in combination with reduced intakes of fat and saturated fatty acids (SFA), are compatible with the improvement of metabolic risk factors for chronic disease, as well as with mean carbohydrate intakes observed in some European countries. This intake range applies to both adults and children older than one year of age.

**Sugars**

Frequent consumption of sugar-containing foods can increase risk of dental caries, especially when oral hygiene and fluoride prophylaxis are insufficient. However, available data do not allow the setting of an upper limit for intake of (added) sugars on the basis of a risk reduction for dental caries, as caries development related to consumption of sucrose and other cariogenic carbohydrates does not depend only on the amount of sugar consumed, but it is also influenced by frequency of consumption, oral hygiene, exposure to fluoride, and various other factors.

The evidence relating high intake of sugars (mainly as added sugars), compared to high intakes of starch, to weight gain is inconsistent for solid foods. However, there is some evidence that high intakes of sugars in the form of sugar-sweetened beverages might contribute to weight gain. The available evidence is insufficient to set an upper limit for intake of (added) sugars based on their effects on body weight.

Observed negative associations between added sugar intake and micronutrient density of the diet are mainly related to patterns of intake of the foods from which added sugars in the diet are derived rather than to intake of added sugars *per se*. The available data are not sufficient to set an upper limit for (added) sugar intake.

Although there is some evidence that high intakes (>20 E%) of sugars may increase serum triglyceride (TG) and cholesterol concentrations, and that >20 to 25 E% might adversely affect glucose and insulin response, the available data are not sufficient to set an upper limit for (added) sugar intake.

Evidence on the relationship between patterns of consumption of sugar-containing foods and dental caries, weight gain and micronutrient intake should be considered when establishing nutrient goals for populations and recommendations for individuals and when developing food-based dietary guidelines.

The Panel notes that a number of authorities have established upper limits for population average intake or individual intake of added sugars of <10 E% but others have not. Typically, such recommendations reflect a judgement of what level of sugar intake is practically achievable within the context of a nutritionally adequate diet based on known patterns of intake of foods and nutrients in specific populations. It is also noted that the average intake of (added) sugars in some EU Member States exceeds 10 E%, especially in children.

**Dietary Fibre**

The role of dietary fibre in bowel function was considered the most suitable criterion for establishing an adequate intake. Based on the available evidence on bowel function, the Panel considers dietary fibre intakes of 25 g per day to be adequate for normal laxation in adults. There is limited evidence to set adequate intakes for children. The Panel considers that the Adequate Intake (AI) for dietary fibre

for children should be based on that for adults with appropriate adjustment for energy intake. A fibre intake of 2 g per MJ is considered adequate for normal laxation in children from the age of one year.

The Panel notes that in adults there is evidence of benefit to health associated with consumption of diets rich in fibre-containing foods at dietary fibre intakes greater than 25 g per day, e.g. reduced risk of coronary heart disease and type 2 diabetes and improved weight maintenance. Such evidence should be considered when developing food-based dietary guidelines.

**Glycaemic index and glycaemic load**

Although there is some experimental evidence that a reduction of the dietary glycaemic index and glycaemic load may have favourable effects on some metabolic risk factors such as serum lipids, the evidence for a role in weight maintenance and prevention of diet-related diseases is inconclusive.

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## BACKGROUND AS PROVIDED BY THE EUROPEAN COMMISSION

The scientific advice on nutrient intakes is important as the basis of Community action in the field of nutrition, for example such advice has in the past been used as the basis of nutrition labelling. The Scientific Committee for Food (SCF) report on nutrient and energy intakes for the European Community dates from 1993. There is a need to review and if necessary to update these earlier recommendations to ensure that the Community action in the area of nutrition is underpinned by the latest scientific advice.

In 1993, the SCF adopted an opinion on the nutrient and energy intakes for the European Community[4]. The report provided Reference Intakes for energy, certain macronutrients and micronutrients, but it did not include certain substances of physiological importance, for example dietary fibre.

Since then new scientific data have become available for some of the nutrients, and scientific advisory bodies in many Eropean Union Member States and in the United States have reported on recommended dietary intakes. For a number of nutrients these newly established (national) recommendations differ from the reference intakes in the SCF (1993) report. Although there is considerable consensus between these newly derived (national) recommendations, differing opinions remain on some of the recommendations. Therefore, there is a need to review the existing EU Reference Intakes in the light of new scientific evidence, and taking into account the more recently reported national recommendations. There is also a need to include dietary components that were not covered in the SCF opinion of 1993, such as dietary fibre, and to consider whether it might be appropriate to establish reference intakes for other (essential) substances with a physiological effect.

In this context the EFSA is requested to consider the existing Population Reference Intakes for energy, micro- and macronutrients and certain other dietary components, to review and complete the SCF recommendations, in the light of new evidence, and in addition advise on a Population Reference Intake for dietary fibre.

For communication of nutrition and healthy eating messages to the public it is generally more appropriate to express recommendations for the intake of individual nutrients or substances in food-based terms. In this context the EFSA is asked to provide assistance on the translation of nutrient based recommendations for a healthy diet into food based recommendations intended for the population as a whole.

## TERMS OF REFERENCE AS PROVIDED BY EUROPEAN COMMISSION

In accordance with Article 29 (1)(a) and Article 31 of Regulation (EC) No. 178/2002, the Commission requests EFSA to review the existing advice of the Scientific Committee for Food on Population Reference Intakes for energy, nutrients and other substances with a nutritional or physiological effect in the context of a balanced diet which, when part of an overall healthy lifestyle, contribute to good health through optimal nutrition.

In the first instance the EFSA is asked to provide advice on energy, macronutrients and dietary fibre. Specifically advice is requested on the following dietary components:

- Carbohydrates, including sugars;
- Fats, including saturated fatty acids, poly-unsaturated fatty acids and mono-unsaturated fatty acids, *trans* fatty acids;
- Protein;

[4] Scientific Committee for Food, Nutrient and energy intakes for the European Community, Reports of the Scientific Committee for Food 31st series, Office for Official Publication of the European Communities, Luxembourg, 1993.

- Dietary fibre.

Following on from the first part of the task, the EFSA is asked to advise on Population Reference Intakes of micronutrients in the diet and, if considered appropriate, other essential substances with a nutritional or physiological effect in the context of a balanced diet which, when part of an overall healthy lifestyle, contribute to good health through optimal nutrition.

Finally, the EFSA is asked to provide guidance on the translation of nutrient based dietary advice into guidance, intended for the European population as a whole, on the contribution of different foods or categories of foods to an overall diet that would help to maintain good health through optimal nutrition (food-based dietary guidelines).

## ASSESSMENT

A draft of this Opinion, agreed by the NDA Panel on 13 March 2009, was published on the EFSA website[5] for public consultation between 5 August and 15 October 2009. The draft Opinion was also discussed at a National Expert Meeting with Member States on Dietary Reference Values held in Barcelona on 7 and 8 September 2009. All the public comments received and comments from Member States that related to the remit of EFSA were assessed and the Opinion has been revised taking relevant comments into consideration. The comments received, a report on the outcome of the public consultation, and the minutes of the meeting with Member States have been published on the EFSA website.

## 1. Introduction

Carbohydrates are the main source of energy in most human diets. Carbohydrates are defined within European legislation (Directive 90/496/EEC) as "metabolisable carbohydrates and including polyols"[6]. Chemically, dietary fibre is also a carbohydrate (EFSA, 2007; Directive 2008/100/EC[7]).

## 2. Definition / category

Chemically, carbohydrates include a range of components such as polyhydroxy aldehydes, ketones, alcohols and acids, as well as their derivatives and polymers, e.g. starch and other polysaccharides. The chemical classification of carbohydrates is usually based on molecular size and monomeric composition, three principal groups being sugars (1–2 monomers), oligosaccharides (3–9 monomers) and polysaccharides (10 or more monomers) (FAO/WHO, 1998, see also Table 1). Due to the chemical diversity of carbohydrates, it is only recently that specific methods for analysis of various carbohydrates in foods have become routinely available. Therefore, carbohydrate values on labels and in food tables are often still derived "by difference" (section 2.1.3).

Nutritionally, it is important to differentiate between two broad categories of carbohydrates: those digested and absorbed in the human small intestine, providing carbohydrates to body cells and those passing to the large intestine, forming substrate for the colonic microflora (Asp, 1996; Englyst and Englyst, 2005). A FAO/WHO Expert Consultation on Carbohydrates in Human Nutrition recommended the introduction of the concept "glycaemic carbohydrate", meaning „providing carbohydrate for metabolism", which corresponds to the previously used term „available carbohydrates" (FAO/WHO, 1998) and to „carbohydrates" according to the European legislation. The nondigestible ("unavailable") carbohydrates are commonly referred to as "dietary fibre" (see 2.1.2 for definitions).

[5] http://www.efsa.europa.eu/EFSA/efsa_locale-1178620753812_1211902045161.htm

[6] Council Directive 90/496/EEC of 24 September 1990 on nutrition labelling for foodstuffs. OJ L 276, 6.10.1990, pp. 40–44.

7 Commission Directive 2008/100/EC of 28 October 2008 amending Council Directive 90/496/EEC on nutrition labelling for foodstuffs as regards recommended daily allowances, energy conversion factors and definitions. OJ L 285, 29.10.2008, pp. 9-12.

**Table 1:** Main types of carbohydrates (Adapted from Asp, 1996).

| Class (DP *) | Sub-group | Components | Monomers | Digestibility** |
|---|---|---|---|---|
| Sugars (1-2) | Monosaccharides | Glucose | | + |
| | | Galactose | | + |
| | | Fructose | | + |
| | Disaccharides | Sucrose | Glu, Fru | + |
| | | Lactose | Glu, Gal | + (–) *** |
| | | Trehalose | Glu | + |
| | | Maltose | Glu | + |
| Oligosaccharides(3-9) | Malto-oligo-saccharfides | Maltodextrins | Glu | + |
| | Other oligo-saccharides | α-Galactosides (GOS) | Gal, Glu | – |
| | | Fructo-oligosaccharides (FOS) | Fru, Glu | – |
| | | Polydextrose | Glu | – |
| | | Resistant dextrins | Glu | – |
| Polyols | Maltitol, sorbitol, xylitol, lactitol | | | +/– |
| Polysaccharides (>9) | Starch | Amylose | Glu | + (–) |
| | | Amylopectin | Glu | + (–) |
| | | Modified starch | Glu | – |
| | | Resistant starch | Glu | – |
| | | Inulin | Fru | – |
| | Non-starch poly-saccharides | Cellulose | Glu | – |
| | | Hemicelluloses | Variable | – |
| | | Pectins | Uronic acids | – |
| | | Other hydrocolloids, e.g. gums, mucilages, β-glucans | Variable | – |
| Related substance | | Lignin | | – |

* DP = Degree of polymerisation
**Denotes digestibility in the small intestine: + digestible, + (–) mainly digestible, +/- partly digestible, – non-digestible
***Lactose is poorly digested by individuals with low intestinal lactase activity
Fru = Fructose, Glu = Glucose, Gal = Galactose

## 2.1. Categories

### 2.1.1. Glycaemic carbohydrates

The glycaemic carbohydrates provide carbohydrate to body cells, mainly in the form of glucose. The main glycaemic carbohydrates are (see also Table 1):

- Glucose and fructose (monosaccharides)
- Sucrose and lactose (disaccharides)
- Malto-oligosaccharides
- Starch (polysaccharide)

In this Opinion the term "sugars" covers monosaccharides and disaccharides. In the literature, various terms are used to differentiate between sugars naturally occurring in foods, e.g. "intrinsic" sugars, and sugars and sugar preparations added to foods, e.g. "added" or "extrinsic" sugars" (IoM, 2005; DoH, 1991). In this opinion the term "added sugars" refers to sucrose, fructose, glucose, starch hydrolysates (glucose syrup, high-fructose syrup) and other isolated sugar preparations used as such or added during food preparation and manufacturing.

Sugar alcohols (polyols) such as sorbitol, xylitol, mannitol, and lactitol, are usually not included in the term "sugars". However, they are partly absorbed and included in "carbohydrates" according to the European legislation.

### 2.1.2. Dietary fibre

The term "dietary fibre" was originally defined as "that portion of food which is derived from cellular walls of plants which are digested very poorly by human beings" (Trowell, 1972). The recognition that polysaccharides added to foods, notably hydrocolloids, could have effects similar to those originating from plant cell walls led to a redefinition of dietary fibre to include "polysaccharides and lignin that are not digested in the human small intestine" (Trowell et al., 1976). The definition and delimitation of "dietary fibre" has been much debated and related both to physiological considerations and to methods that can be used for dietary fibre analysis in foods (FAO/WHO, 1998; Asp, 1995 and 1996; Englyst and Hudson, 1996; Englyst and Englyst, 2005; Englyst et al., 2007).

Non-starch polysaccharides (NSP) are the main constituents of dietary fibre and include a host of different polymers, highly variable in terms of molecular size and structure, as well as in monomeric composition. Main classes of non-starch polysaccharides are cellulose, hemicelluloses, pectins, and other hydrocolloids. Due to the structural variability, different non-starch polysaccharides may have very different physical-chemical properties, which are of key importance for their physiological effects. For example cellulose is insoluble in water, whereas pectins and hydrocolloids, e.g. guar gum and mucilages, may form highly viscous water solutions. Resistant starch is insoluble and indigestible due to its physical form or enclosure in cellular structures, whereas resistant oligosaccharides are readily soluble in water but do not form viscous solutions. The terms "soluble" and "insoluble" dietary fibre have been used in the literature to differentiate between viscous, soluble types of fibre (e.g. pectins) and insoluble components such as cellulose. The distinction was mainly based on the different physiological effects. However, this differentiation is method-dependent, and solubility does not always predict physiological effects. Therefore, FAO/WHO proposed the distinction between soluble and insoluble fibre should be phased out (FAO/WHO, 1998).

The interest in defining and quantifying dietary fibre in foods lies in the physiological effects that are associated with their consumption, which include decreased intestinal transit time and increased stool bulk, reducing blood total and/or LDL cholesterol concentrations, and reducing post-prandial blood glucose and /or insulin concentrations, among others (AFSSA 2002; NNR, 2004; IoM, 2005; GR, 2006; Mann et al., 2007). These physiological effects of dietary fibre are distinct from those of glycaemic carbohydrates.

In national and international recommendations on dietary fibre intake, the definitions are generally in accordance with and related to analysis with methods approved by the Association of Official Analytical Chemists (AOAC). Definitions differ with respect to some minor components such as fibre of animal origin and some synthetic or isolated fibre constituents (Annex 1).

The U.S. Food and Nutrition Board (FNB) defines "total dietary fiber" as the sum of "dietary fiber", consisting of non-digestible carbohydrates and lignin that are intrinsic and intact in plants, and "functional fiber", consisting of isolated, non-digestible carbohydrate components with demonstrated beneficial physiological effects in humans (IoM, 2005). The rationale behind this differentiation is that there is epidemiological evidence for beneficial effects of foods naturally high in dietary fibre, such as whole-grain cereals, some fruits and vegetables, and that dietary fibre can be regarded as a marker of such foods. The argument that the term "dietary fibre" should be restricted to non-starch polysaccharides of cell wall origin (Englyst and Englyst, 2005; Englyst et al., 2007) has a similar rationale. Consequently, according to the FNB, documentation of the beneficial effects of added, functional fibre is required for inclusion in "total dietary fibre".

The Panel notes that a major problem in making this differentiation in practice is that analytical methods cannot differentiate between "dietary fibre" and "functional fibre" once they occur mixed in a food product,

and similarly NSP from plant cell walls cannot be differentiated from added NSP with similar monomeric composition.

In view of the key importance of small-intestinal digestibility for the nutritional effects of carbohydrates, the Panel considers that dietary fibre should include all non-digestible carbohydrates. This includes non-starch polysaccharides, resistant starch, resistant oligosaccharides with three or more monomeric units and other non-digestible, but quantitatively minor components that are associated with the dietary fibre polysaccharides, especially lignin (Cho et al., 1997; AACC, 2001; AFSSA, 2002; NNR, 2004; GR, 2006). This definition is in accordance with the definition brought to step 8 in the Codex Alimentarius (Codex, 2009) and agreed by the Codex Alimentarius Committee in 2009, although the inclusion of non-digestible carbohydrates with 3 to 9 monomeric residues is so far left to the national authorities. As in the EU definition, beneficial physiological effects have to be demonstrated before addition of natural or synthetic fibre to foods (Annex 1).

The minimum chain length of three monomeric units, degree of polymeration (DP) 3, was set since undigestible oligosaccharides with DP3-9, such as fruco-oligosaccharides (FOS) and galacto-oligosaccharides (GOS) are natural constituents of many foods, quantitatively important in e.g. artichokes and beans, respectively. There is considerable evidence of "fibre-like" effects of these oligosaccharides, such as promoting a presumably "healthy" microflora, short-chain fatty acids (SCFA)-production in the colon, and enhancement of calcium absorption from the colon. Undigestible disaccharides are not prominent constituents of normal foods and not well characterised physiologically. However, if such ingredients will be available in the future and shown to have "fibre-like" effects, there may be reasons to reconsider the DP3 limit.

For the purpose of this Opinion, dietary fibre is defined as non-digestible carbohydrates plus lignin. The Panel considers that the main types of total dietary fibre are:

- Non-starch polysaccharides (NSP) - cellulose, hemicelluloses, pectins, hydrocolloids (i.e. gums, mucilages, β-glucans).
- Resistant oligosaccharides - fructo-oligosaccharides (FOS), galacto-oligosaccharides (GOS), other resistant oligosaccharides.
- Resistant starch - consisting of physically enclosed starch, some types of raw starch granules, retrograded amylose, chemically and/or physically modified starches.
- Lignin associated with the dietary fibre polysaccharides.

*Methods of analysis*

Current enzymatic gravimetric or enzymatic chemical methods for dietary fibre cover NSP, analytically resistant starch and lignin. However, given that dietary fibre is a mixture of chemically heterogeneous carbohydrate components, several analytical methods are currently required to measure all fractions of dietary fibre. Methods measuring NSP alone (Englyst and Hudson, 1996) give lower estimates than methods for total dietary fibre in foods containing resistant starch, and/or lignin, e.g. whole-grain flour and cereals processed in a way that generates resistant starch. On the other hand, methods determining dietary fibre, including resistant starch, measure the fraction, which includes mainly retrograded amylose, resistant to the enzymes used in the assay. Finally, resistant oligosaccharides and inulin are not included in any of the current methods for total dietary fibre, and therefore need to be measured separately and subsequently added to the total fibre estimate (Cho et al., 1997; Champ et al., 2001 and 2003).

### 2.1.3. Total carbohydrates

In many food composition tables and in food labelling, carbohydrates are still expressed “by difference”, which means that moisture, protein, fat and ash are analytically determined and the rest named

"carbohydrates". This non-specific procedure includes all kinds of carbohydrates regardless of their physiological and nutritional properties, as well as variable amounts of non-carbohydrate material, e.g. organic acids, lignin and polyphenols (FAO/WHO, 1998; Southgate, 1995; Asp, 1995).

## 2.2. Metabolism

### 2.2.1. Glycaemic carbohydrates

The glycaemic carbohydrates provide carbohydrates to body cells, mainly in the form of glucose. In general, only monosaccharides are absorbed in the small intestine. The enzymatic degradation of starch begins by the action of salivary amylase and is continued in the small intestine by pancreatic amylase. The degradation products – mainly maltose and oligosaccharides – are hydrolysed further to glucose by a set of enzymes, "disaccharidases", bound to the brush border membrane of the enterocytes. The same enzymes hydrolyse the dietary disaccharides. Glucose and galactose are absorbed efficiently by a secondary active carrier coupled with sodium (sodium glucose transporter 1, SGLT1), whereas fructose is absorbed by facilitated diffusion that does not involve sodium co-transport (GLUT5). The absorption of monosaccharides is regarded as the rate-limiting step.

Absorbed monosaccharides are transported to the liver and then to the systemic circulation. The cellular uptake is mediated by a number of glucose transporters (GLUT1–4), variously expressed in different tissues. Insulin is a key hormone for the uptake and metabolism of glucose. The plasma insulin concentration increases immediately after ingestion of glycaemic carbohydrates. Unlike glucose, fructose enters body cells without the need for insulin. The metabolism of fructose, therefore, favours lipogenesis more than glucose. In liver cells, fructose is phosphorylated to fructose-1-phosphate that can be converted to fatty acids, providing a route for lipogenesis in addition to that shared with glucose (via glucose/fructose-6-phosphate) (Vasankari and Vasankari, 2006). Both fructose and galactose, the latter arising from hydrolysis of lactose, are also transformed to glucose mainly in the liver.

### 2.2.2. Glycaemic index and glycaemic load

The concept of glycaemic index (GI) was introduced by Jenkins and co-workers in 1981, in order to rank foods in a standardised way according to their effects on blood glucose levels after a meal. The FAO/WHO Expert Consultation defined GI as the incremental area under the blood glucose response curve during 1.5–3 hours after intake of a 50 g carbohydrate portion of a test food, and expressed as a percentage of the response to the same amount of carbohydrate from a standard food taken by the same subject (FAO/WHO, 1998). Glucose or white bread is used as standard. GI values obtained with the white bread standard are typically about 40% higher than those obtained with the glucose standard which is the generally preferred standard. GI values for about 750 foods have been published (Foster-Powell et al., 2002) and recently updated with additional data to contain 2480 individual food items (Atkinson et al., 2008).

Whereas it was previously assumed that sugars are rapidly absorbed and therefore have a higher GI than polysaccharides (e.g. starch), which are slowly absorbed, a number of food-related factors have been identified to determine the GI. For instance, fructose has a low GI (30 with the white bread reference as 100) and sucrose an intermediate GI, i.e. lower than white bread (Björck et al., 2000). Starchy foods, on the other hand, can have low, intermediate or high GI, depending on their composition (amylose/amylopectin ratio) and physical/chemical state. The swelling and dissolution of starch at wet heat treatment, known as gelatinisation, is particularly important in making starch more readily accessible to digestive enzymes. Physical barriers such as in intact cereal grains, cellular structures in leguminous seeds, parboiled rice and whole fruits, and the protein network in pasta products, are food-related factors lowering the GI. Organic acids (acetic, propionic and lactic acid) decrease the glycaemic response to foods or meals, mainly due to inhibition of gastric emptying (Liljeberg and Björck, 1998). Viscous, soluble types of dietary fibre may also delay gastric emptying, in addition to their inhibitory effect on diffusion and transport in the small intestine (Brown et al., 1999; Jenkins et al., 2000).

In practice, the blood glucose response after a meal is influenced by both the GI and the amount of carbohydrate in a portion of a food. Consequently, the glycaemic load (GL) concept was introduced in 1997 to quantify the glycaemic effect of a portion of food (Salmeron et al., 1997a and 1997b). GL is defined as the amount of glycaemic carbohydrate in a food times the GI of the food/100, and the sum of individual GL values for foods and meals has been used to estimate the glycaemic load of the whole diet.

Studies have shown that the glycaemic response to a meal can be predicted from properly determined GI of the constituent foods (Wolever et al., 2006; Wolever and Jenkins, 1986; Järvi et al., 1999). However, the glycaemic response can also be influenced by the protein and fat content, and by the type and amount of beverage taken with the food (Henry et al., 2006). Flint et al. (2004) found no correlations between predicted postprandial glucose responses based on published GI values for foods and the measured glucose elevations after different breakfast meals containing 50 g available carbohydrates and varying amounts of fat and protein. This indicates that the composition and the size of a meal, i.e. in terms of energy and macronutrient content, are also important determinants for the glycaemic responses. Validated GI values for food products are needed in studies investigating effects of GI.

### 2.2.3. Dietary fibre

The components included in dietary fibre are by definition resistant to hydrolysis and absorption in the small intestine. They pass the upper gastro-intestinal tract and enter the colon substantially unmodified. Viscous, water-soluble fibre such as β-glucans and pectin can modify blood glucose response and total and LDL-cholesterol concentrations by interfering with digestion and absorption of glycaemic carbohydrates and cholesterol and/or bile acids, respectively. Inhibitory effects on mineral absorption, i.e. of iron, zinc and calcium, have been attributed to fibre-associated complexing compounds, notably phytic acid in cereals and leguminous seeds.

Dietary fibre components are subject to more or less extensive anaerobic fermentation by the colonic microflora. The extent of fermentation is dependent on both substrate and host factors, e.g. molecular structure and physical form of the substrate, bacterial flora and transit time. Less fermentable types of fibre, such as in lignified outer layers of cereal grain, generally have the most prominent faecal bulking effects due to their ability to bind water in the distal colon. Fermentable fibre also contributes to the faecal bulk through increased microbial mass.

Some fermentation products, such as propionic acid and butyrate, may influence also systemic metabolism, i.e. cholesterol synthesis and possibly insulin sensitivity. Fermentable dietary fibre components, including oligosaccharides that are often referred to as “prebiotics”, increase Bifidobacteria and Lactobacilli which produce lactate and short-chain fatty acids such as acetate, propionate and butyrate (Gibson and Roberfroid, 1995). These fatty acids inhibit the fermentation of protein components, which could produce potentially toxic products, especially ammonia and amines. Short-chain fatty acids decrease the pH of the colonic content, which stimulates colonic absorption of minerals, notably calcium, and inhibits formation of potential co-carcinogens from bile acids. Butyrate is a main source of energy for the colonic mucosa and has effects on cell differentiation and apoptosis with possible implications for colon carcinogenesis. Acetate and propionate are absorbed from the colon and thus provide energy to the host (Cummings et al., 2004).

The absorption of fermentation products, i.e. short chain fatty acids, means that dietary fibre contributes to the energy content of the diet, but less than glycaemic carbohydrates. The contribution is variable depending on the extent of fermentation. FAO/WHO (1998) has recommended the use of an average energy factor for dietary fibre, 8 kJ or 2 kcal per g, and this recommendation has been now included in the EU nutrition labelling Directive[8].

[8] Council Directive of 24 September 1990 on nutrition labelling for foodstuffs (90/496/ECC). OJ L 276, 6.10.1990, p.40 ammended by Commission Directive 2008/100/EC of 28 October 2008.

# 3. Dietary sources and intake data

## 3.1. Dietary sources

### 3.1.1. Glycaemic carbohydrates

Main dietary sources of glucose and fructose are fruits, berries, fruit juices and some vegetables. Free galactose is rare in foods, except in fermented and lactase-hydrolysed milk products. Fruits, berries and juices are natural sources of sucrose, although sugar added to foods, carbonated beverages and sweets or in the household usually provides most of the dietary sucrose. More or less completely hydrolysed starch or high fructose syrup, in which about half the glucose is isomerised to fructose, are increasingly used in some countries, to replace sucrose in confectionary and carbonated drinks.

Lactose occurs exclusively in milk and milk products. Human milk has the highest lactose content of all milks, 7 g per 100 g. The lactose content in cow‟s milk is around 5 g per 100 g. Digestible malto-oligosaccharides originate mainly from partly hydrolysed starch.

Main dietary sources of starch are bread and other cereal products, potatoes, tubers and pulses (FAO/WHO, 1998).

### 3.1.2. Dietary fibre

Whole grain cereals, pulses, fruit and vegetables and potatoes are the main sources of dietary fibre. Also nuts and seeds contain high concentrations. Cellulose occurs together with hemicelluloses in cereals. The lignified outer layers are the predominant fibre source in whole-grain products. Oats and barley contain high concentrations of a water-soluble, viscous type of polysaccharide, β-glucan. Pectins, a main type of dietary fibre in fruits and vegetables, have similar properties.

## 3.2. Dietary intake

Typical intakes of carbohydrates and dietary fibre are presented for children and adolescents in 19 countries (Annex 2a and 2b) and for adults in 22 countries in Europe (Annex 3a and 3b). The data refer to individual based food consumption surveys, conducted from 1994 onwards. Most studies comprise national representative population samples. The data were derived from national reports and from a recently published overview (Elmadfa, 2009).

As shown in Annexes 2 and 3, there is a large diversity in the methodology used to assess individual intakes of children, adolescents and adults. Because the different methods apply to different time frames, this inevitably resulted in variance in both the quality and quantity of available data, which make direct comparability difficult. Moreover, age classifications are in general not uniform. Comparability might also be hampered by differences in food composition tables used for the conversion of food consumption data to estimated nutrient intakes (Deharveng et al., 1999). Food consumption data are prone to reporting errors and there might be a varying degree of underreporting in different surveys.

Although these differences may have an impact on the accuracy of between country comparisons and the results should be interpreted with caution, the presented data give a rough overview of the carbohydrate intake in a number of European countries. Most studies reported mean intakes and standard deviations (SD) or mean intakes and intake distributions. In most studies the contribution of carbohydrates to energy intake is based on total energy intake (including the energy from alcohol).

### 3.2.1. Total carbohydrates

Average carbohydrate intakes in children and adolescents in European countries varied between 41 to 58 E% (Annex 2b). Most of the reported average intakes (82%) were between 47 and 55 E%; approximately 13% were above 55%. Within population ranges varied from 38 to 49 E% (5$^{th}$ percentile) to 63 to 66 E% (95$^{th}$ percentile).

In adults average carbohydrate intakes varied from approximately 38 to 54 E% (Annex 3b). In the various age categories average intake of carbohydrates ranged from approximately 41 to 51 E% (19-34 years), 38 to 49 E% (35 to 64 years) and 40 to 53 E% (65 years and over), respectively. More than half (53%) of the reported mean values were between 45 and 50 E%; Average carbohydrate intakes of 50 E% and higher were achieved in 16% of the adult groups belonging to the age categories 19 to 34 years and 65 years and over. Within population ranges varied from 31 to 34 E% at the lower (5$^{th}$ percentile) to 58 to 61 E% at the upper end (95$^{th}$ percentile) of the distributions. Mean intakes were highest in the Czech Republic and Norway and lowest in Greece and Spain.

As shown in Annexes 2b and 3b, not all countries reported intakes of mono-, disaccharides and polysaccharides. When reported, average intakes of mono- and disaccharides varied between 23 to 36 E% in children and adolescents, with highest intakes in infants, whereas the intake of polysaccharides was between 23 and 25 E%.

In Finnish infants aged 8 to 13 months the reported average intake of sucrose was 3 to 5 E%. This amount increased in children aged 2 to3 years to approximately 10 to 12 E%. In schoolchildren and adolescents average intakes varied between 11 and 25 E%. More than half (56%) of these average intakes were between 10 and 15 E%. Available intake distributions showed that five percent of the children and adolescents had average intakes of 20 E% and above.

In adults the intake of mono and disaccharides varied between 17 to 26 E% and the intake of polysaccharides between 20 to 27 E%.

Average sucrose intake in adults varied from 6 to nearly 14 E%. Average intakes below 11 E% were only observed in the older age categories (35 to 64 years: in 94%; 65 years and over: in 79% of the group). Within population ranges varied from 1 to 4 E% at the lower (5$^{th}$ percentile) to 17 to 25 E% at the upper end (95$^{th}$ percentile) of the distribution.

### 3.2.2. Dietary fibre

Apart from infants and young children, average dietary fibre intakes varied between 10 to 20 g per day in young children (<10 to 12 years), and from 15 to 33 g per day in adolescents. The highest intakes were observed in German adolescents (Annex 2b). Within population ranges varied from 6 to 8 g per day (5$^{th}$ percentile) to 25 to 46 g per day (95$^{th}$ percentile). Expressed per MJ reported intakes were between 1.7 g per MJ and 2.5 g per MJ. Studies on German children followed from 6 months up to 18 years of age (data not presented) show that the energy adjusted fibre intake was highest at 1 year (3 g per MJ), thereafter declining to about 2.5 g per MJ in preschool- and school-age (Alexy et al., 2006).

In adults average dietary fibre intakes ranged from 15 to 30 g per day. For subjects aged 65 years and over, about 70% of the reported intakes were between 19 and 25 g per day. In the other age categories these percentages were 44% (19-35 years) and 42% (50 to 64 years), respectively. Within population ranges varied from 6 to 9 g per day (5$^{th}$ percentile) to 39-51 g per day (95$^{th}$ percentile). A few countries presented (also) the intake of dietary fibre per MJ. Then daily intakes ranged from 1.6 to 3.6 g per MJ in adults.

## 4. Overview of dietary reference values and recommendations

A number of national and international organisations have set dietary reference values (DRVs) for carbohydrates (total and/or glycaemic) as well as for dietary fibre (Table 2). Generally, reference intakes are expressed as percent of the total energy intake (E%). For fibre, intakes are expressed in grams per day and/or on an energy basis (per MJ or per 1,000 kcal).

### 4.1. Glycaemic carbohydrates

According to the Nordic Nutrition Recommendations (NNR, 2004) carbohydrates (including energy from dietary fibre, 8 kJ per g) should provide 50 to 60% of the total energy intake. The population goal is 55 E% from carbohydrates, which should be used for planning purposes. The intake of refined, added sugars should not exceed 10 E%. Although not explicitly stated in the report, it appears that this is a recommendation for individuals. Refined sugars include sucrose, fructose, glucose, starch hydrolysates (glucose syrup, high-fructose syrup) and other isolated sugar preparations used as such or added during food preparation and manufacturing. The basis for the recommendation on added sugars is to ensure an adequate intake of essential nutrients and dietary fibre, especially in children and older adults with a low energy intake.

The Health Council of the Netherlands (GR, 2001) based their recommendations for digestible carbohydrates on the 97.5 percentile of the endogenous production of glucose. At this level of carbohydrate intake the breakdown of tissue protein is minimal. Using data on average glucose production and assuming a coefficient of variation (CV) of 20%, DRVs were established for the various age and sex groups. The recommendations for glycaemic carbohydrates for adults (19 to 70 years) were calculated to be 272 to 282 g per day, corresponding to about 40 E%. For children and adolescents, recommendations are 45 to 50 E%. No upper limit for digestible carbohydrates is given. No quantitative recommendation was made for (added) sugars. The Netherlands guidelines for healthy eating did not specify recommended values for (added) sugars consistent with adequate nutrition and the prevention of chronic diseases because insufficient scientific evidence was available to support firm conclusions (GR, 2006).

In the Nutritional Recommendations for the French Population (AFSSA, 2001), the Reference Intake for total carbohydrates was set at 50 to 55 E%. It is stated that the reference intake is a consequence of the recommendations for fat (30 to 35 E%) and protein (8 to 10 E%). It is also stated that presently no carbohydrate constituents indispensable for growth and maintenance, which cannot be synthesised by humans, have been identified. Other considerations include e.g. energy density of the diet and effects on serum lipids. A carbohydrate intake above 55 E% is stated to be associated with risk of dyslipidemia (e.g. increased VLDL- and decreased HDL-cholesterol). Various considerations are given with respect to the contribution of MUFA and carbohydrates for reducing cardiovascular risk, treatment of obesity and the metabolic syndrome. Carbohydrates and MUFA should together contribute two-thirds of the total energy intake, with a range for carbohydrates of 40 to 55 E%. No quantitative recommendation was made for (added) sugars.

In the German-Austrian-Swiss recommendations (D-A-CH, 2008), the guiding value ("Richtwert") for carbohydrate intake is at least 50 E%. This value applies to populations and is based on evidence from epidemiological studies, and studies linking a high intake of (saturated) fat with cardiovascular risk factors and other diseases. It is stressed that carbohydrates should be derived from foods rich in starch and dietary fibre, and that intake of refined sugars should be limited. In order to avoid gluconeogenesis from protein (e.g. amino acids) and to inhibit lipolysis at least 25% of the energy should be supplied from carbohydrates. This percentage applies to all ages. No quantitative recommendation was made for (added) sugars.

WHO gives population nutrient intake goals (population average intakes that are judged to be consistent with the maintenance of health in a population) for preventing diet-related chronic diseases (WHO/FAO, 2003). For carbohydrates the population goal is set at 55-75 E%, including dietary fibre. This goal is the percentage of energy available after taking into account the proportion recommended as protein and fat. A recent FAO/WHO Scientific Update on carbohydrate in the human diet proposes that the range is extended

to 50 to 75 E% (Mann et al., 2007). It was proposed that the population average intake of free sugars, defined as "all monosaccharides and disaccharides added to foods, by cook or consumer, plus sugars naturally present in honey, syrups and fruit juice", should not exceed 10 E%. The basis for this goal is that high intakes of free sugars are associated with decreased nutrient density, and risk of weight gain, especially when consumed as beverages.

The UK Committee on Medical Aspects of Food Policy (DoH, 1991) set a dietary reference value (population average intake) for starches and intrinsic and milk sugars of 37 E%. This figure was based on considerations that starch and intrinsic sugars should provide the balance of dietary energy not provided by those nutrients for which the intake should be restricted, i.e. alcohol, protein, fat and non-milk extrinsic sugars. The reference value is applicable to adults and children above 2 years of age. For non-milk extrinsic sugars the population"s average intake should not exceed 60 g per day or 10 E%, based mainly on the role of frequent consumption of such sugars in dental caries.

The US Food and Nutrition Board estimated the average requirement of (glycaemic) carbohydrates as 100 g per day for children and adolescents up to 18 years, as well as adults (IoM, 2005), based mainly on data regarding glucose utilisation by the brain. The RDA was set at 130 g per day, assuming a CV of 15%. The RDA corresponds to about 18 and 25 E% in adult males and females, respectively, assuming an energy intake of 2,800 and 2,100 kcal per day, respectively. The US Food and Nutrition Board also set Acceptable Macronutrient Distribution Ranges (AMDR) for total carbohydrates of 45 to 65 E% for individuals. The AMDR are based on evidence indicating a decreased risk for coronary heart disease (CHD) at low intakes of fat and high intakes of carbohydrates, and on evidence for an increased risk of obesity and its complications, including CHD, with high intakes of fat. For added sugars, although there were insufficient data to set a UL (e.g lack of evidence on dose-response for dental caries), a maximal intake level of 25 E% or less from added sugars for individuals was proposed to prevent the displacement of foods that are major sources of essential micronutrients.

**Table 2:** Recommended dietary intakes for adults.

| | USA[a] (IoM, 2005) | Nordic Countries (NNR, 2004) | WHO (2003) | Netherlands (GR, 2001 and 2006) | France, (AFSSA, 2001) | Germany, Austria, Switzerland (D-A-CH, 2008) | Eurodiet (2000) | UK (DoH, 1991) |
|---|---|---|---|---|---|---|---|---|
| Protein, E% | 10-35 | 10-20 | 10-15 | 8-11 | 8-10 | 10-11 | - | 9 |
| Fat, E% | 20-35 | 25-35 | 15-30 | 20-40<br>20-30/35[b] | 30-35 | 30 | < 30 | 33 |
| Carbohydrates, total, E% | 45-65 | 50-60 | 55-75 | 40[c] | 50-55 | > 50 | > 55 | 47[d] |
| Sugars, E% | < 25[e] | < 10[e] | < 10[f] | - | - | | < 4 occasions per day[g] | <10[h] |
| Dietary fibre, g/day | w: 25<br>m: 38 | 25-35 | > 25[i] | 32-45 | 25-30 | 30 | > 25 | 18[j] |
| g/MJ | 3.4 | 3 | | 3.4 | | W: 3<br>M: 2.4 | 3 | - |

(a) AMDR: acceptable macronutrient distribution ranges, applies to individuals. AI for dietary fibre
(b) For subjects with BMI >25 or with undesireable weight gain
(c) RDA for digestible carbohydrates
(d) Intrinsic and milk sugars and starch 37 E%
(e) Refined, added sugars include sucrose, fructose, glucose, starch hydrolysates (glucose syrup, high-fructose syrup) and other isolated sugar preparations used as such or added during food preparation and manufacturing
(f) Free sugars, defined as all monosaccharides and disaccharides added to foods, plus sugars naturally present in honey, syrups and fruit juice
(g) Corresponds to an intake of < 10 E%
(h) Non-milk extrinsic sugars
(i) Total dietary fibre from wholegrain cereals, fruit and vegetables, 20 g NSP
(j) Refers to non-starch polysaccharides

### 4.2. Dietary fibre

The Health Council of the Netherlands (GR, 2006) has set guidelines for the intake of dietary fibre mainly based on the importance of dietary fibre for intestinal function and its relationship to risk of coronary heart disease. For children a gradual increase in the intake is recommended from 2.8 g per MJ at 1 to 3 years of age, 3.0 g per MJ at 4 to 8 years of age, 3.2 g per MJ at 9 to 13 years of age to 3.4 g per MJ from 14 years of age. An upper limit is not specified.

In the Nordic Nutrition Recommendations, the recommended intake of dietary fibre is set to 25 to 35 g per day in adults, i.e. approximately 3 g per MJ (NNR, 2004). An Adequate Intake of dietary fibre reduces the risk of constipation and can most likely contribute to protection against colon cancer. No recommendation is given for children due to limited evidence, but it is stated that intake of appropriate amounts of dietary fibre from a variety of foods is important for children as well and that from school age the intake should gradually increase during adolescence to reach the recommended level for adults.

In the WHO report (2003) there is no precise population goal for the intake of total dietary fibre, but at least 25 g per day should be provided from fruit, vegetables and whole-grain foods. This population goal is based on evidence linking high intake of dietary fibre (from fruit, vegetables and whole-grain foods) with decreased risk of e.g. weight gain (convincing), diabetes type 2 (probable), cardiovascular diseases (probable). The food-based recommendation was supported by the recent FAO/WHO Scientific Update on carbohydrate in the human diet (Mann et al., 2007).

In the Nutritional Recommendations for the French Population (AFSSA, 2001), an intake of dietary fibre of above 25 g per day is recommended for maintaining "a healthy colon" and to decrease the risk of colon cancer, with 30 g per day as a preferred level. Also, an increased intake of dietary fibre is stated to be advantageous in conditions such as dyslipidaemia and in diabetes mellitus type 2.

The guiding value for dietary fibre in the German-Austrian-Swiss recommendations is at least 30 g per day, corresponding to about 3 g per MJ for women and 2.4 g per MJ for men, respectively (D-A-CH, 2008). The basis for the value are studies associating increased dietary fibre intake with decreased risk of constipation, diverticulosis, colon cancer, gallstone formation, overweight, hypercholesterolaemia, diabetes mellitus type 2 and atherosclerosis.

The UK Committee on Medical Aspects of Food Policy (DoH, 1991) set a dietary reference value for non-starch polysaccharides (NSP) to 18 g per day, with an individual range of 12 to 24 g per day. The value is based on the effect of non-starch polysaccharides on bowel function and stool weight. The reference value refers to adults and is not applicable to children. An upper limit was set at 32 g per day.

The U.S. Food and Nutrition Board (IoM, 2005) set an Adequate Intake (AI) for total dietary fibre of 3.4 g per MJ (14 g per 1,000 kcal) based on the energy-adjusted median intake associated with the lowest risk of CHD in observational studies. The AI is applicable for all age and sex categories from 1 year of age. The AI corresponds to 25 g per day for women and 38 g per day for men aged 14 to 50 years, respectively.

ESPGHAN (European Society for Pediatric Gastroenterology Hepatology and Nutrition) concluded that by school age an otherwise balanced diet is likely to provide at least 10 g per day of dietary fibre, and that the intake should then gradually increase to reach the recommended level for adults during adolescence. An intake of dietary fibre too high might be a cause of inadequate energy and nutrient density to cover the needs of small children (Aggett et al., 2003).

## 5. Criteria (endpoints) on which to base the dietary reference values

The amount and type of carbohydrates and dietary fibre in the diet may affect both short-term and long-term metabolic responses such as serum lipids or plasma glucose and insulin concentrations, which can be

regarded as candidate criteria for establishing DRVs. Serum LDL-cholesterol has been causally related to the risk of developing cardiovascular diseases (EFSA, 2004; IoM, 2005), while serum triglycerides, LDL/HDL ratio or total cholesterol/HDL ratio have also been associated with cardiovascular disease (CVD) risk in epidemiological studies (EFSA, 2004; Austin et al., 1998). In addition to cardiovascular diseases, other long-term endpoints for establishing DRVs for both glycaemic carbohydrates and dietary fibre include body weight control, gastrointestinal function, diabetes and some cancers.

The DRVs apply to healthy populations and they are not intended as reference values for the treatment of patients with diseases or conditions like diabetes, obesity, or CVD. However, they apply to healthy subjects with signs of metabolic disturbances like impaired glucose tolerance, elevated blood pressure, serum lipids, etc.

Apart from carbohydrates and dietary fibre, the amount and type of fat and protein in the diet also influence these metabolic factors. As energy balance is the ultimate goal, it is necessary to consider macronutrients in combination and, as a consequence, on an energy basis (percent of energy intake, E%).

## 5.1. Total glycaemic carbohydrates

Most of the studies addressing the effects of macronutrient manipulation on health outcomes do not allow a precise differentiation between total and glycaemic carbohydrates due to limitations in the food composition data, and thus dietary intake data. However, glycaemic carbohydrates generally contribute 90 to 95% of the total energy derived from carbohydrate intake.

### 5.1.1. Dietary requirements

Glucose is a preferred energy source for most body cells, and can be stored as glycogen in the liver and in the muscles. The storage capacity is limited, in total to around 500 g for an adult, of which 300 to 400 g can be stored in the muscles. Liver glycogen is essential for liver functions such as detoxification by conjugation with glucuronic acid, and is used to maintain normal blood glucose concentrations between meals. Muscle glycogen is used primarily as a source of energy within the muscles.

Only cells in the central nervous system, red blood cells and some other cells dependent on anaerobic glycolysis have an absolute requirement for glucose (IoM, 2005). The body can synthesise glucose from protein and glycerol. Provided that the diet contains adequate amounts of protein (i.e. amino acids) and glycerol (as e.g. triglycerides) for *de novo* synthesis of glucose, it has been generally assumed that there is no need for dietary carbohydrates (IoM, 2005). Feeding studies in pregnant rats and dogs have, however, shown that diets devoid of carbohydrates can induce still birth, but also high mortality of the offspring and low birth weight (Romsos et al., 1981; Koski et al. 1986: Koski and Hill, 1986).

After a prolonged deficit of glucose, brain cells can adapt partially to utilise fat-derived metabolites, i.e. β-hydroxybutyric acid and acetoacetic acid. A very low carbohydrate diet, providing less than around 50 g per day, however, results in a chronically increased production and plasma concentrations of these acids, referred to as ketosis. Absence of glycogen stores has adverse effects on high-intensity energy production by muscles (Hultman et al., 1999). An intake of 50 to 100 g per day of glycaemic carbohydrates generally prevents ketosis.

In practice, diets totally devoid of carbohydrates have probably not been consumed by any population group during evolution. The diets of Greenland Eskimos and Alaskan Inuits have been reported to contain from 3 to 53 E% carbohydrates; current intakes are >40 E% (Jokelainen, 1965; Bang et al., 1980; Nobmann et al., 2005).

Ketogenic diets with very low carbohydrate contents (from 4 to 5 to 10 E%) have been used as an alternative therapy in children with epilepsy not responding to drug therapy and are a mandatory therapy for some inborn errors of metabolism (e.g. pyruvate dehydrogenase deficiency) (Keene, 2006; Klepper and Voit,

2002; Wexler et al., 1997; Vining, 1999). A number of adverse effects related to such diets have been reported including constipation, nutritional deficiencies and deaths (Wheless, 2001; Papandreou et al., 2006). Severe acidosis is another reported adverse effects of diets very low in carbohydrates consumed by adults who wanted to loose weight (IoM, 2005; Shah and Isley, 2006; Chen et al., 2006a). Methylglyoxal and its by-products accumulate during ketosis and are recognised as a potential cause of blood vessel and tissue damage (Beisswenger et al., 2005).

In conclusion, an intake of 50 to 100 g glycaemic carbohydrates per day is sufficient to avoid ketosis and 130 g per day for both children (> 1 year) and adults is estimated to cover the needs of glucose for the brain (IoM, 2005). However, these levels of intake are not sufficient to meet enegy needs in the context of acceptable intake levels of fat and protein.

### 5.1.2. Glucose tolerance and insulin sensitivity

The blood glucose concentration is determined by three main factors: the rate of intestinal carbohydrate uptake, the net liver uptake or release (from gluconeogenesis and glycogenolysis), and the peripheral glucose uptake, which is in turn dependent upon the insulin level and the peripheral insulin sensitivity/resistance. With a constant dietary carbohydrate load, there is a range of blood glucose responses between individuals, from low responses with a continuum to what is defined as impaired glucose tolerance (IGT) and type-2 diabetes. Physical activity has the potential to enhance insulin sensitivity and thereby decrease the glycaemic response to a meal (Borghouts and Keizer, 2000; Ivy, 1997).

Some small scale and short-term intervention studies designed to assess the effects of high carbohydrate (50 to 85%E), low fat (<25E%) vs low carbohydrate (8 to 40%E), high fat (>35%) intakes on measures of glucose tolerance or insulin sensitivity suggest that high carbohydrate intakes may improve insulin sensitivity and/or glucose tolerance both in non-diabetic and diabetic subjects (McClenaghan, 2005), although the available data are not consistent (IoM, 2005).

There are no long-term studies published specifically designed to address the impact of macronutrient (i.e. fat/carbohydrate) manipulations on glucose intolerance or insulin sensitivity under isocaloric conditions in adults. *Ad libitum* fat-reduced diets leading to higher carbohydrate intakes (from 46 E% to 55 E%) have been shown to improve glucose tolerance in IGT subjects in the context of significant weight loss (about 3kg) after one year (Swinburn et al., 2001; Mensink et al., 2003). However, no conclusions can be drawn from these studies regarding the effects of carbohydrate consumption itself, independent of weight loss, on glucose tolerance.

The same argument applies to epidemiological studies associating high fat, low carbohydrate intakes, to decreased glucose tolerance or insulin sensitivity. The extent to which those observations are confounded by body weight and/or body fat gain has not been fully elucidated (IoM, 2005).

In conclusion, and although the influence of dietary carbohydrates on glucose tolerance and insulin sensitivity is still unclear, total carbohydrate intakes of 46 to 55 E% appear to be compatible with the maintenance of a normal glucose tolerance and insulin sensitivity in healthy subjects and in subjects with signs of the metabolic syndrome.

### 5.1.3. Serum lipids

The effects of dietary variation in total carbohydrate intakes on LDL-cholesterol are strongly associated with the type of fat which is replaced by carbohydrates. When saturated fatty acids are kept constant, varying carbohydrate intake as a function of total fat has no effects on LDL-cholesterol concentrations (IoM, 2005).

Data from intervention studies consistently show that increasing carbohydrate intakes (in the range of about 30 to 70 E%) as an inverse function of fat (ranging from 50 to 18 E% as fat) at low intakes of saturated fatty acids (SFA, <10%E) induces a decrease in plasma concentrations of HDL-cholesterol and an increase in the

total/HDL-cholesterol ratio and TG concentrations (Sacks and Katan, 2002; EFSA, 2004; IoM, 2005). This effect is largely attenuated in the lean and physically active (IoM, 2005). Also, TG concentrations are consistently higher when SFA are replaced by carbohydrates rather than by monounsaturated fatty acids (MUFA), particularly in hypertrygliceridaemic subjects (Aro et al., 1998; Mensink et al. 2003; Appel et al. 2005; Berglund et al., 2007; Furtado et al., 2008), and the HDL-cholesterol lowering effect is more pronounced in subjects with higher HDL concentrations at baseline (Obarzanek et al., 2001). However, even if TG concentrations consistently increase with increasing carbohydrate intakes when administered in isocaloric conditions, this effect could be attenuated when carbohydrate-rich diets are consumed *ad libitum*, possibly due to a concomitant reduction in body weight (Rock et al., 2004; Retzlaff et al., 1995; Kasim-Karakas et al., 2000).

In conclusion, the adverse effects of increasing total carbohydrate intakes on the lipid profile provide a basis to set an upper bound of RI for total carbohydrates.

### 5.1.4. Body weight

The impact of macronutrient manipulation on body weight in the context of weight management in overweight and obese subjects may be different from the effects on prevention of weight gain in leaner persons and may depend on whether diets are administered *ad libitum* or under isocaloric conditions.

In intervention trials tightly controlling energy intake, energy expenditure, weight loss and weight maintenance are a function of energy intake rather than of the macronutrient composition of the diet (Poppitt et al., 2002; IoM, 2005; van Dam and Seidell, 2007; Nordman et al., 2006; Sacks et al., 2009). However, even when an equivalent energy intake is intended, fat-reduced diets (<35 E%) tend to be hypocaloric compared with carbohydrate-reduced diets (<50 E%) and increase long-term compliance with energy restriction, leading to slightly greater weight loss (IoM, 2005; van Dam and Seidell, 2007).

Several randomised intervention studies suggest that fat-reduced (< 25 to 30 E%), moderately high carbohydrate (> 50 E%) diets consumed *ad libitum* have the potential to prevent weight gain in normal weight subjects and produce weight loss in overweight (BMI > 25kg/m$^2$) individuals as compared to higher fat (> 35 E%), lower carbohydrate (40 to 50 E%) diets (IoM, 2005). However, although very-low carbohydrate diets (< 40 E%) consumed *ad libitum* may have an advantage in terms of weight loss up to 1 year over lower (< 35 E%) and very-low (10 E%) fat diets (van Dam and Seidell, 2007; Gardner et al., 2007; Shai et al., 2008), long-term weight regain tends to be higher and may not offer clear benefits in terms of long-term body weight control (van Dam and Seidell, 2007).

In some long-term (>1 year) intervention studies, dietary modifications with a shift from a habitual Western-type, relatively high fat (35 to 40 E%), moderately low carbohydrate (40 to 50 E%) diets to more carbohydrate-rich (>50 E%), fat-reduced (<30 E%) diets consumed *ad libitum* were reported to be associated with a reduced risk of weight gain or a moderate weight loss in various population groups including normal, overweight and obese subjects (IoM, 2005; Howard et al., 2006a; Lanza et al., 2001).

Results from prospective cohort studies are conflicting with respect to the relationship between carbohydrate intake and weight gain (Halkjær et al., 2006; Gaesser, 2007).

In conclusion, the adverse effects associated with high fat, low carbohydrate diets on short- and long-term body weight control provide a basis to set a lower bound of the RI for total carbohydrates.

### 5.1.5. Type 2 diabetes mellitus

There are no intervention studies specifically addressing the effect of carbohydrate intake on the risk of developing type-2 diabetes. In two lifestyle, long-term intervention studies including weight loss by reducing fat intake and increasing physical activity, the presumed increase in carbohydrate intake (targeted at 55 E% carbohydrates) was compatible with a lower risk for type 2 diabetes mellitus (Tuomilehto et al.

2001, Knowler et al., 2002; Lindström et al., 2003, 2006a and 2006b). Large-scale observational studies on the effects of carbohydrate intake and risk of diabetes have yielded conflicting results (IoM, 2005).

Several cohort studies have investigated the relationship between intake of total and individual carbohydrates and the risk of developing diabetes type 2 (Murakami et al., 2005; McKeown et al., 2004; Meyer et al., 2000). Generally, no or weak relations between total carbohydrate intake and diabetes risk were observed.

In conclusion, diets providing about 55 E% as carbohydrates are compatible with a lower risk for type 2 diabetes mellitus in the context of concomitant weight loss and physical activity.

### 5.1.6. Cardiovascular disease

As reviewed (Sacks and Katan, 2002), three dietary intervention studies reducing total fat intake, in particular saturated fat, and increasing the consumption of carbohydrate-rich foods, did not significantly reduce the risk of cardiovascular disease. However, it cannot be excluded that the duration, compliance, and sample sizes may have been insufficient to produce a reduction in coronary events. Also, in the Women's Health Initiative Dietary Modification Trial a dietary intervention that reduced total fat intake and increased intakes of carbohydrates from vegetables, fruits, and grains did not significantly reduce the risk of cardiovascular disease (Howard et al., 2006b).

Data from observational studies, and in particular from the U.S. Nurses Health Study, do not indicate any consistent relationship between total carbohydrate intake and CHD risk (Liu et al., 2000; Oh et al., 2005; Halton et al., 2006). Data from one cohort (Halton et al., 2006) showed that an increased carbohydrate intake was associated with an increased risk of total and haemorrhagic stroke in women with a BMI > 25 kg/m$^2$ (Oh et al., 2005). Conversely, in two large prospective cohort studies (Trichopoulou et al., 2007; Lagiou et al., 2007), low energy-adjusted carbohydrate intakes, particularly if combined with high protein intakes, were associated with a significantly higher risk of mortality from CVD.

In conclusion, data from intervention and observational studies do not show any consistent relationship between the intake of total or glycaemic carbohydrate intake and the risk of CVD. The ranges of carbohydrate intakes in the studies above vary from 30 to 70 E%.

## 5.2. Sugars

### 5.2.1. Nutrient density of diet

Nutrient density is the amount of nutrients in foods per unit of energy. An adequate nutrient density is essential for providing recommended intakes of nutrients, especially in individuals with a low energy intake. There is some evidence that high intakes of added sugars, particularly from low nutrient density foods, might be associated with a decrease in the nutrient density of the diet („nutrient dilution") due to displacement of nutrient rich foods (van Dam and Seidell, 2007). In some EU countries, studies in children and elderly nursing home residents (Lyhne and Ovesen, 1999; Beck and Ovesen, 2002; Alexy et al., 2003a; Øverby et al., 2004; Kranz et al., 2005; Frary et al., 2004) have shown that an intake of >10 to 30 E% of added sugars (mono-, disaccharides and higher saccharides) is associated with a reduced intake of several micronutrients (e.g. calcium, iron, folate, vitamin A) and dietary fibre, especially in children and adults with energy intakes below about 8 MJ per day. Data from the U.S. indicate that nutrient density of the diet among children was negatively correlated to the intake of added sugars in the range of 10 to 25 E% or above, but that clear differences were seen mainly at very high intakes (>25 E%) (IoM, 2005), with some exceptions e.g. calcium in preschool children (Kranz et al., 2005). A systematic review of 15 cross-sectional studies comprising children and adults shows that there are insufficient and conflicting data with respect to the relation between intake of added sugars and nutrient density, with no clear evidence of micronutrient dilution or a threshold for a quantitative amount of added sugar intake for any of the micronutrients

investigated (Rennie and Livingstone, 2007). The observed inverse association of nutrient density or intake with added sugar intake may be partly explained by methodological issues, e.g. different definitions of added sugars, and confounding effects related to differences in energy intake (higher sugar intake as E% in individuals with lower total energy intake and consequently lower nutrient intake). The association between added sugar intake and micronutrient density of the diet is mainly dependent on the intake patterns of the food groups from which added sugars in the diet are derived (Rennie and Livingstone, 2007).

In conclusion, observed negative associations between added sugar intake and micronutrient density of the diet are mainly related to patterns of intake of the foods from which added sugars in the diet are derived rather than to intake of added sugars *per se*. The available data are not sufficient to set an upper limit for (added) sugar intake. Evidence on the relationship of foods containing added sugar to micronutrient density of the diet and to micronutrient intake of population groups should be considered when developing food-based dietary guidelines.

### 5.2.2. Glucose tolerance and insulin sensitivity

Some mainly small, short-term (4 to 6 weeks) studies have investigated the effect of sugar intake on glucose and insulin response comparing individual sugars (sucrose, glucose or fructose) or mixtures of different sugars with starch or "complex carbohydrates". The majority of these used iso-caloric diets aimed at body weight maintenance during the study. Both normal subjects and subjects with impaired insulin responses were included. The amount of sugars in the intervention diets varied from about 3 to10 E% in the "low-sugar" diets to 20 to 30 E% in the high-sugar diets. The studies are summarised in Annex 4. Two of the three studies with sucrose showed increased insulin concentrations at sucrose intakes of 18 and 33 E%, whereas one did not show any difference between diets providing 10 or 25 E% sucrose. One study showed increased glucose concentrations at sucrose intakes of 18 and 33 E%, whereas one did not show any difference between diets providing 10 or 25 E% sucrose.

In conclusion, there are limited, and mainly short-term, data on the effects of high intakes of sugars on glucose and insulin response. Most studies do not find any adverse effects at intakes of predominantly added sugars up to 20 to 25 E%, provided that body weight is maintained.

### 5.2.3. Serum lipids

A number of mainly small, short-term (2 to 6 weeks) studies have investigated the effect of sugar intake on serum lipids, comparing individual sugars (sucrose, glucose or fructose) or mixtures of different sugars with starch or "complex carbohydrates". The majority of studies used iso-caloric diets aiming at body weight maintenance during the study. Both normal subjects and subjects with impaired insulin responses were included. The amount of sugars in the intervention diets varied from about 3 to 10 E% in "low-sugar" diets to 20 to 30 E% in the high-sugar diets. The studies are summarised in Annex 5. In five of the seven studies, increased sugar intakes led to increases in total and LDL-cholesterol, in four, the TG-concentrations increased, but responses differed according to sex and insulin sensitivity. Effects on HDL-cholesterol were less prominent or not reported. Overall, negative effects were observed at sugar intakes > 20 E%. Only in the study by Hallfrisch et al. (1983) a significant increase in total, LDL-cholesterol and TG was seen at fructose intakes of 7.5 and 15 E%. Effects tended to be more pronounced in subjects with markers of metabolic syndrome, e.g. insulin resistance. Studies also varied with respect to the composition of the basic and intervention diets, which might have influenced the results. For example Black et al. (2006) found that a high-sucrose diet (25 E%) resulted in increases in total and LDL cholesterol by 15% and 24%, respectively, compared to the control diet (10 E% sucrose). The authors hypothesise that the higher level of SFA and lower level of PUFA in the high-sucrose diet could have contributed to the cholesterol-raising effect.

Few long-term studies of the effect of sugars on lipids have been published. In a six-month study by Saris et al. (2000), 316 obese subjects were randomised to three groups, which received either a control diet with 46 E% carbohydrates of which 24 E% was starch and 21 E% sugars, or an intervention diet with 52 to 56 E%

carbohydrates containing mainly starch (33 E% starch, 16 E% sugars) or mainly sugars (sucrose, fructose and lactose, 30 E%). All diets were given *ad libitum*. No significant changes were seen in serum lipids among the groups.

In a sub-study 46 obese subjects with the metabolic syndrome (≥3 risk factors) were randomised to receive either one of three diets *ad libitum*: a control diet with with 29 E% starch and 21 E% sugars, two fat-reduced diets, one with 53 E% carbohydrates, mainly as "complex carbohydrates" (33 E% starch and 18 E% sugars) and one high-sugar diet with 57 E% carbohydrates including 29 E% sugars (sucrose, fructose and lactose) (Poppitt et al., 2002). Thirty-nine subjects completed the study. After six months fasting serum TG was higher in the sugar group than in the "complex-carbohydrate" and control groups, respectively. Weight loss was correlated with a decrease in TG concentrations.

Smith et al. (1996) found that restriction of the intake of added sugars during six months led to reduced TG concentrations in hyper-triglyceridaemic, overweight subjects, even at relatively moderate intakes (from about 12 to 4 E% added sugars). This reduction was partly associated with an (unintentional) weight-loss of <2% of the initial body weight.

In conclusion, a number of small-size controlled, iso-caloric short-term studies (2 to 6 weeks), indicate that high intakes (>20 E%) of sugars, provided predominantly as added sucrose or fructose, may increase serum TG and LDL-cholesterol concentrations, especially in subjects with markers of the metabolic syndrome, e.g. insulin resistance. However, data on dose-response are limited, especially at intakes in the range of 5 to 20 E%, albeit one study found elevated lipid concentrations at a fructose intake of 7.5 E%. Information on the intake of total sugars is lacking in some studies. In long-term intervention studies in which diets were given *ad libitum* (<6 months) changes in blood lipids as result of diets high in sugars (about 30% E) or of sugar restriction (from 12 to 4 E%) are closely related to body weight changes. A number of dietary factors such as fatty acid composition, dietary fibre content and type may modulate the effects. There are insufficient data to set a UL for sugars based on their effects on serum lipids.

### 5.2.4. Other cardiovascular risk factors

In the study by Marckmann et al. (2000) nonfasting FVIIc (factor VII coagulant activity) was lower on the low-sucrose (2.5 E%) diet compared to the high-sucrose (23 E%) diet.

In a 10-week intervention study, overweight subjects (mean BMI 28kg/m$^2$) were given 1.3 L sucrose-sweetened or artificially sweetened soft drinks per day while otherwise eating an *ad libitum* habitual diet (Raben et al., 2002). Those subjects consuming the sugar-sweetened soft drink had a sucrose intake of 28 E% and increased their energy intake during the study. At the end of the study blood pressure was increased in this group (SBP +3.8, DBP +4.1 mmHg), while it was decreased among subjects who consumed the artificially sweetened soft drink (SBP –3.1, DBP -1.2 mmHg). Body weight (+1.6 kg) and fat mass (+1.3 kg) increased in the sugar-group, with no significant changes in the control group.

In conclusion, there are insufficient data to set a UL for sugars based on their effects on the risk factors for cardiovascular disease reported in this section.

### 5.2.5. Body weight

The evidence relating high intake of sugars (mainly as added sugars), compared to high intakes of starch, to weight gain is inconsistent (IOM, 2005; van Dam and Seidell, 2007). Either weight loss (Saris et al., 2000) or weight maintenance (Poppitt et al., 2002) has been reported for high carbohydrate (52 to 56 E%), high-sugar (29 to 30 E%) diets as compared to control diets (49 E% as carbohydrates, 21 E% sugars) consumed *ad libitum* for six months. Epidemiological studies do not show a positive correlation between total sugar intake and obesity – rather the opposite (IoM, 2005).

There is some evidence that sugar sweetened beverages do not induce satiety to the same extent as solid forms of carbohydrate, and that high intakes of sugars in the form of sugar-sweetened beverages might contribute to weight gain (van Dam and Seidell, 2007; Mann *et al*., 2007). *Ad libitum* consumption of high sucrose diets (28 E% mainly as beverages) was found to increase body weight and fat mass as compared to lower sucrose diets with artificial sweeteners (Raben et al., 2002). In a systematic review Malik et al. (2006) included 30 studies, mainly in children and adolescents (15 cross-sectional, 10 prospective, and 5 experimental), that investigated the association between sugar-sweetened beverage intake and weight gain. The authors state that large cross-sectional studies and well-powered prospective cohort studies with long periods of follow-up show a positive association between higher intakes of sugar-sweetened beverages and weight gain and obesity in both children and adults. No data on overall effect size were included. Vartanian et al (2007) included 88 studies in a meta-analysis regarding the association between soft drink consumption and body weight. Most studies were cross-sectional (17) and longitudinal (10) and included both children and adults. The overall effect size across studies was 0.08 (expressed as change in BMI and/or body weight) ($p<0.001$). However, results from another meta-analysis of eight prospective studies and two intervention studies among children and adolescents did not show a clear quantitative relationship and suggested that there may be publication bias against studies that do not report statistically significant findings (Forshee et al., 2008). Long-term randomized controlled trials on the effects of sugar sweetened beverages on body weight are lacking (van Dam and Seidell, 2007; Johnson et al., 2009).

Fructose has been suggested to play a specific role in weight gain (Elliot et al., 2002, see 2.2.1). There are, however, few and mainly short-term controlled intervention studies in healthy subjects comparing fructose with other sugars or carbohydrate-sources and these do not allow a conclusion regarding the role of fructose in obesity (Vasankari and Vasankari, 2006).

In conclusion, the evidence relating high intake of sugars (mainly as added sugars), compared to high intakes of starch, to weight gain is inconsistent for solid foods. However, there is some evidence that high intakes of sugars in the form of sugar-sweetened beverages might contribute to weight gain. The available evidence is insufficient to set an upper limit for sugars based on their effects on body weight. Evidence on the relationship of sugar-sweetened beverages and body weight should be considered when developing food-based dietary guidelines.

### 5.2.6. Type 2 diabetes

Evidence on the effects of sugar consumption on the risk of developing type 2 diabetes comes primarily from large prospective cohort studies. No (Colditz et al., 1992; Janket et al., 2003) or even inverse (Meyer et al., 2000) associations have been reported for total sugars and/or specific types of sugars and diabetes risk. However, consumption of sugar-sweetened beverages, and particularly if sweetened with glucose or fructose, was found to be positively associated with increased type 2 diabetes risk (Schultze et al., 2004a; Montonen et al., 2007). The available evidence is insufficient to set a UL for sugars based on their effects on type 2 diabetes risk.

### 5.2.7. Dental caries

Increased risk of dental caries in children is associated with a high frequency (more than about 4 times daily) of intake of cariogenic sugars (mainly sucrose, glucose, and fructose) rather than with the total amount of dietary sugars; the evidence indicates that frequent consumption of sweets and confectionery products and sugar-containing drinks is associated with a higher risk of caries (Moynihan and Petersen, 2004; DoH, 1991; IoM, 2005; Anderson et al., 2009).

Caries develops as tooth substance demineralises upon pH decrease due to fermentation of carbohydrates by tooth-colonising bacteria into different organic acids. Dental caries is an infectious disease, although sucrose and other easily fermentable sugars, e.g. glucose and fructose, play a key role (Navia, 1994; Lingstrom et al., 1997). Foods rich in starch may also contribute, especially when the starch molecule is easily available to

degradation by amylase. The acid production from lactose in dental plaque is normally low, while the fermentation of starch varies greatly and depends on the degree of gelatinisation (Lingstrom et al, 1997). Decreases in pH to well below 5.5 are considered critical for caries development in enamel (the tooth crown). In tooth roots the critical pH for demineralisation is approximately 6.5. In addition to lactic acid, sucrose fermentation produces insoluble extracellular glucose polymers leading to voluminous biofilms that favour colonisation of cariogenic streptococci on the teeth surfaces.

Dental caries prevalence has declined in many European countries during the last decades of the 20th century, but trends vary between countries and age groups (Touger-Decker and van Loveren, 2003; Schulte et al., 2006; Haugejorden and Magne Birkeland, 2006; Demertzi et al., 2006; Stecksen-Blicks et al., 2008; Pitts et al., 2006).

More recently, mainly cross-sectional studies generally find a weak or moderately strong relationship between the intake of sucrose and other sugars and caries prevalence (Burt and Pai, 2001). The impact of fluoride prophylaxis and other lifestyle variables seems to override variations in cariogenic carbohydrate intake in these studies. High intake of sugars has been associated with an increased risk of caries when oral hygiene is simultaneously poor and at a low level of fluoride prophylaxis (Danish Nutrition Council, 2003; Burt and Pai, 2001; Kleemoja-Kujala and Räsänen, 1982). However, results from a Finnish longitudinal study suggest a relationship between the intake of sucrose and sucrose containing foods and caries development during childhood among children with fluoride prophylaxis (Karjalainen et al., 2001; Ruottinen et al., 2004).

Available data do not allow the setting of an UL for sugars on the basis of a risk reduction for dental caries, as caries development related to consumption of sucrose and other cariogenic carbohydrates does not depend only on the amount of sugar consumed, but it is also influenced by various other lifestyle factors (oral hygiene, exposure to fluoride, meal frequency and diet composition), heredity, illness, medication, malnutrition, and flow and composition of saliva.

In conclusion, frequent consumption of sugar-containing foods can increase risk of dental caries, especially when prophylactic measures, e.g. oral hygiene and fluoride prophylaxis, are insufficient. However, available data do not allow the setting of an UL for sugars on the basis of a risk reduction for dental caries, as caries development related to consumption of sucrose and other cariogenic carbohydrates does not depend only on the amount of sugar consumed, but it is also influenced by oral hygiene, exposure to fluoride, frequency of consumption, and various other factors. Evidence on the relationship of frequency of consumption of sugar-containing foods and dental caries should be considered when developing food-based dietary guidelines.

## 5.3. Dietary fibre

### 5.3.1. Dietary requirements

Dietary fibre has not been shown to be an indispensable component of the diet. However, dietary fibre has a major role in bowel function and gastro-intestinal symptoms, such as constipation (IoM, 2005).

### 5.3.2. Gastrointestinal function

#### 5.3.2.1. Adults

Dietary fibre has a major role in bowel function and gastro-intestinal symptoms, such as constipation, have been linked to low fibre intakes (IoM, 2005). Constipation has been defined as difficulty in passing stools or an incomplete or infrequent passage of hard stools (Longstreth et al, 2006). Constipation occurs in 5 to 18%

of adults in different countries with a greater percent of women and elderly affected and adversely affects the quality of life (Wald et al., 2008). It may also contribute to diverticular disease.

Both observational and experimental data show that dietary fibre is the most important dietary determinant of faecal bulk and transit time (Cummings et al., 1992, Birkett et al., 1997). Dietary fibre from cereals, fruits, and vegetables increases stool weight, which promotes normal laxation in children and adults. In general, the greater the weight of the stool and the more rapid the rate of passage through the colon the better the laxative effect (Birkett et al., 1997).

It has also been demonstrated that different kinds of dietary fibre have different bulking capacity. Dietary fibre in wheat bran and other fibre that is fairly resistant to fermentation in the large bowel has the most pronounced bulking effect (5 to 6 g per g dietary fibre) mainly due to water binding in the distal bowel, whereas more fermentable fibre provide some bulk mainly due to increased bacterial mass (Cummings, 2001).

Although there is no single accepted definition of what constitutes normal laxation, frequency of defaecation is typically about once per day on Western diets (Weaver, 1988). Haack et al. (1998) have indicated that a defecation frequency of about once per day and a transit time in the range of 2 to 3 days may be considered normal laxation. They reported that while increasing intake of fibre (provided by a mixture of fruit, vegetables, and grains) from 16 to 30 g per day increased defaecation frequency from 0.7 to 0.94 times per day, a further increase in fibre intake to 42 g per day had no further effect on defecation frequency, which remained at about once per day. There was no significant change over the range of fibre intakes in gastrointestinal transit time, which remained within the range of 2 to 3 days, or stool moisture, which remained within the range of 70 to 74%. Faecal weight increased from 109 g to 156 g and 195 g at fibre intakes of 16, 30 and 42 g per day, respectively.

Increasing dietary fibre intake from 12 to 45 g per day increased faecal weight from 69 to 184 g per day and reduced transit time from >70 h to 45 h (Stasse-Wolthuis et al., 1978). Mean transit time in UK adults has been reported as 70 h (median 60 h) (Cummings et al., 1992) in a population in which mean stool weight is about 110 g per day, with weights of less than 100 g per day in about 50% of individuals, and dietary fibre intake of about 18 g per day (Cummings et al., 1992). It has been estimated that a dietary fibre intake of 25 g per day is associated with stool weight of about 130 to 150 g per day (Cummings et al., 1992). Birkett et al. (1997) reported that adults who consumed 25 g dietary fibre in their usual diet excreted more than 150 g faeces per day.

Taken together, these data indicate that an intake of 25 g per day of dietary fibre from mixed foods (as AOAC fibre or equivalent) is compatible with an intestinal transit time of about two to three days and a defaecation frequency of 1 per day and a faecal moisture of >70% and may be considered adequate for normal laxation in adults.

#### 5.3.2.2. Children

There is evidence that constipation is a common problem also during childhood (e.g. Loening-Baucke, 1993) and that there is an inverse relationship with dietary fibre intake (Edwards and Parrett, 2003).

There are few data relating intake of dietary fibre to normal laxation in children. Data from long-term intervention and observational studies can, however, give information on fibre intakes that are compatible with adequate growth and development and at the same time provide pre-requisites for good health. Results from the Finnish STRIP-study indicate that a fibre intake corresponding to 2-2.5 g per MJ is compatible with normal growth and development. The fibre intake among German children was already at one year somewhat higher (3 g per MJ) and there are no reports of adverse effects related to the fibre intake.

In conclusion, dietary fibre intake of 2 g per MJ should be adequate for normal laxation in children based on the dietary fibre intake that is considered adequate for normal laxation in adults (25 g, equivalent to 2 to 3 g

per MJ for daily energy intakes of 8 to 12 MJ) and taking into account that energy intake relative to body size in children is higher than in adults.

### 5.3.3. Glucose tolerance and insulin sensitivity

Few intervention studies have investigated the effects of fibre intake on measures of glucose tolerance or insulin sensitivity.

In the two-year intervention study by Mensink et al. (2003) conducted in subjects with impaired glucose tolerance (IGT), glucose tolerance improved in the intervention group compared to the control group. Dietary fibre intake was 3.1 to 3.3 g per MJ in the intervention group compared to 2.7 g per MJ in the control group.

A number of cohort studies have found that intake of fibre and fibre rich foods such as wholegrain cereals correlated favourably with measures of glucose tolerance or insulin sensitivity. Liese et al. (2005) found that dietary fibre intake was associated positively with insulin sensitivity and inversely with fasting insulin, but not with acute insulin response, in 979 adults with normal or impaired glucose tolerance. In the baseline data of an intervention study by Lau et al. (2005) the intake of dietary fibre was inversely associated with the probability of having insulin resistance (assessed by the homeostasis model assessment of insulin resistance, HOMA-IR) among middle-aged, healthy adults. Similar results were observed in another cross-sectional study on subjects at high risk of type 2 diabetes (relatives of patients with type 2 diabetes) (Ylönen et al., 2003).

In conclusion, increasing intakes of foods rich in dietary fibre are associated with reduced risk of impaired glucose control. Dietary fibre intakes associated with favourable effects are >2.6 g per MJ and about 30 g per day, although the contribution of dietary fibre *per se* to this effect remains to be established.

### 5.3.4. Serum lipids

A meta-analysis including 67 intervention studies showed that intakes of 2 to 10 g per day of viscous, soluble fibre (e.g. pectin, oat bran, guar gum, psyllium) were associated with a small, but significant, decrease in total cholesterol (-0.045 mmol/L per gram of dietary fibre) and LDL-cholesterol (-0.057 mmol/L per gram) (Brown et al., 1999). The effects have been confirmed in subsequent studies in both hypercholesterolaemic subjects (Jenkins et al., 2002), and normocholesterolaemic subjects (Berg et al., 2003; Aller et al., 2004), but not in the study by Chen et al. (2006b). Controlled intervention studies have generally shown that fasting TG concentrations are not affected by fibre intake (Queenan et al., 2007; Beer et al., 1995; Behall et al., 2004; Anderson et al., 1995; Braaten et al., 1994; van Horn et al., 1991).

Certain kinds of fibre, especially soluble, viscous types, can, however, reduce post-prandial hyperlipidaemia (Lairon, 2001). These effects are related to diminished cholesterol and/or bile acid absorption (Andersson, 1996) and possibly also to products of colonic fermentation. Effects on lipid metabolism of resistant starch and resistant oligosaccharides demonstrated in experimental animals have so far not been reproduced in man.

In conclusion, viscous types of dietary fibre may contribute to reducing total and LDL-cholesterol concentrations. The effects are limited at amounts usually consumed from foods.

### 5.3.5. Blood pressure

In a meta-analysis Whelton et al. (2005) evaluated 25 randomised controlled trials with respect to the effect of dietary fibre intake on blood pressure. The difference in fibre intake between intervention and control groups ranged from 3.8 to 12.5 g per day, with a median difference of 10.7 g per day. In eight studies fibre was given as a supplement, otherwise fibre was provided as foods (15 studies, cereal, fruit, fruit/vegetables, cereal/fruit, cereal/vegetables/fruit), pectin (1 study) or guar gum (1 study). Study duration varied from 2 to

26 weeks. Overall, dietary fibre intake was associated with a small, but significant, reduction in diastolic blood pressure (-1.6 mmHg) and a non-significant reduction in systolic BP (-1.1 mmHg). A significant reduction in both systolic and diastolic blood pressure was observed in trials conducted in patients with hypertension (SBP -5.9 mmHg, DBP -4.2 mmHg) and in trials including both normotensive and hypertensive subjects with a duration of the intervention of 8 weeks or longer (SBP -3.1 mmHg, DBP -2.6 mmHg). A further meta-analysis by Streppel et al. (2005) showed similar results. Whether the effect is related to the intake of dietary fibre *per se*, to the consumption of other nutrients in fibre-rich food products with blood-pressure lowering effects, or both, was not addressed.

In conclusion, small, but rather consistent, effects on blood pressure have been observed for diets rich in fibre from e.g. cereals, fruit and vegetables, although the contribution of dietary fibre *per se* to this effect remains to be established.

### 5.3.6. Body weight

#### 5.3.6.1. Adults

Reviews of randomised trials have shown weight loss in a majority of studies with no differences between fibre types or between fibre occurring in foods or in supplements (Pereira and Ludwig, 2001; Howarth et al., 2001). This led to the conclusion by the World Health Organisation (WHO) that the evidence for a protective effect against weight gain and obesity of high dietary intake of NSP (dietary fibre) was convincing (WHO/FAO, 2003) and that an intake of at least 25 g total dietary fibre per day from wholegrain cereals, fruit and vegetables would be desireable. Results from seven prospective cohort studies show an inverse relationship between weight gain and baseline intake or change in fibre intake among adults during follow-up periods up to 12 years (Lairon, 2007; Koh-Banerjee et al., 2004). However, Iqbal et al. (2006) found no significant relationship between fibre intake at baseline and subsequent weight change during a 5-year follow-up among 30 to 60 year old men and women.

In the Finnish Diabetes Prevention study an increase in dietary fibre intake was associated with a sustained weight reduction (>5%) (Lindström et al., 2006a). The odds ratio for sustained weight loss at year 3 of the study (1 to 3 year follow up) was 2.04 (95% CI: 1.05 to 3.95) for subjects in the third quartile (3.1 to 3.7 g per MJ) and 2.67 (95%CI: 1.26 to 5.65) for subjects with a fibre intake in the upper quartile (>3.7 g per MJ), compared to subjects in the lowest quartile (< 2.6 g per MJ).

#### 5.3.6.2. Children

In the Finnish intervention study STRIP (Special Turku Coronary Risk Factor Intervention Project) children were given dietary advice from the age of 7 to 8 months and have been followed up to 14 years of age (Niinikoski et al., 2007). These children grew and developed normally (Kaitosaari et al., 2003). Data on fibre intake are available for children up to 7 years of age. Mean intake of fibre varied from 9.2 g per day at 13 months of age to 11.8 g per day at 5 years of age (Lagström et al., 1999). The energy adjusted fibre intake was between 1.9 and 2.5 g per MJ at 13 months of age, 1.8 and 2.3 g per MJ at 3 years of age and 1.7 and 2.4 g per MJ at 5 years of age. There were no differences in body weight or growth in relation to fibre intake. At 7 years of age fibre intake was, depending on dietary pattern, between 12.3 and 15.5 g per day, corresponding to 1.9 and 2.4 g per MJ, respectively (Räsänen et al., 2002).

Studies on children eating mixed diets do not indicate adverse effects on growth due to high fibre intake. On the other hand, there are studies indicating that dietary fibre intake can contribute to lower the risk of obesity (Edwards and Parrett, 2003).

In conclusion, increased intake of dietary fibre, both from naturally fibre-rich foods and added fibre or fibre supplements, has been shown to be related to improved weight maintenance in adults and sustained weight reduction in overweight subjects. Estimated intakes associated with this effect in adults are in the order of

>25 g dietary fibre per day (from wholegrain cereals, fruit and vegetables) and >3.1 g total fibre per MJ. Results of intervention and observational studies in children indicate that a fibre intake corresponding to 2 to 3 g per MJ is compatible with normal growth and development.

### 5.3.7. Colorectal cancer

The effect of dietary fibre on faecal bulk has been an important parameter in setting recommended dietary intakes for dietary fibre. Intake in adults necessary to obtain a faecal bulk related to a minimal risk of intestinal disorders, particularly colon cancer, has been estimated to be 26-34 g per day (Cummings et al.,1992), 35 to 45 g per day (Spiller and Spiller, 2001) and 32 to 40 g per day (Monro, 2004).

The fermentation of dietary fibre by the colonic microflora has been recognised as important for colonic health and might have systemic metabolic effects through absorption of fermentation products. One of the fermentation products, butyric acid, is of special interest in relation to colon cancer since it is a main source of energy for colonocytes and has effects on cell differentiation, apoptosis and inflammatory processes (Cummings et al., 2004).

A large number of both *in vitro* and *in vivo* studies have provided mechanistic support for protective effects of dietary fibre against colon cancer (e.g., effects on faecal enzymes, secondary co-carcinogenic bile salt metabolites, etc.). Furthermore, there is a relationship between fibre intake and faecal bulk, and further between colon cancer and faecal bulk (Cummings et al., 1992; Birkett et al., 1997). Stool outputs of 150 g per day or more, normally obtained at a dietary fibre intake of at least 25 g per day, have been associated with lower prevalence of colon cancer (Cummings et al., 1992 and 2004).

A number of mainly small-size clinical intervention studies have been performed using polyp recurrence and rectal cell proliferation as surrogate markers for colon cancer (IoM, 2005). A pooled analysis of two larger intervention studies comprising 3,209 subjects with colorectal adenomas showed that increased intakes of fibre were associated with a significantly decreased risk of adenoma recurrence among men, but not in women, after 3 to 4 years (Jacobs et al., 2006). However, the strategy to increase fibre intake differed between studies (wheat bran supplement vs dietary intervention aimed at decreasing fat intake and increasing intake of fibre, fruit and vegetables).

Recent epidemiological studies have, however, given inconsistent results regarding a protective effect of dietary fibre against colorectal cancer (WCRF/AICR, 2007; Otani et al., 2006; Park et al., 2005; Bingham et al., 2003 and 2005). In the EPIC (European Prospective Investigation into Cancer and Nutrition) study comprising more than 0.5 million people from ten different European countries, dietary fibre intake from foods was inversely related to the incidence of large bowel cancer with an adjusted relative risk of 0.75 (95% CI 0.59 to 0.95) for the highest versus the lowest quintile of intake and 0.58 (0.41 to 0.85) after adjustment for more detailed dietary data. The association with colon cancer was strengthened with longer follow-up. However, the association with rectal cancer was no longer significant (Bingham et al., 2005).

In the World Cancer Research Fund/American Institute of Cancer Research (WCRF/AICR) report 16 cohort studies were reviewed and of these, eight could be included in a meta-analysis (WCRF/AICR, 2007). The analysis showed a statistically significant risk reduction of colorectal cancer and the overall relative risk was 0.90 (95% CI: 0.84 to 0.97) for a 10 g per day increase in fibre intake. However, a previous pooled analysis of 13 prospective cohort studies did not show a statistically significant protective effect after adjusting for known risk factors (Park et al., 2005). The WCRF/AICR concluded that foods containing dietary fibre might protect against colorectal cancer, albeit residual confounding in the studies could not be excluded.

In a nested case-control study Peters et al. (2003) found that high intakes of dietary fibre were associated with a lower risk of colorectal adenomas (polyps), especially regarding fibre from grains and cereals and from fruits.

In conclusion, dietary fibre is the most important dietary factor for faecal bulk and regular bowel movements, and might reduce the risk of colon cancer. Previous estimates of fibre intakes in adults necessary to achieve minimal risk of intestinal disorders, particularly colon cancer, range from 26 to 45 g per day. One meta-analysis of prospective cohort studies found a 10% decrease in the risk of colorectal cancer for each 10 g per day increase of dietary fibre intake. However, a consistent relation has not been shown in all cohort studies.

#### 5.3.8. Type 2 diabetes mellitus

In the Finnish Diabetes Prevention Study an increase in dietary fibre intake was associated with a reduced risk of developing diabetes type 2 in subjects with IGT (Lindström et al. 2006b). The adjusted hazards ratios were 0.50 (95% CI: 0.28 to 0.89), 0.71 (0.40 to 1.23) and 0.38 (0.19 to 0.77) for subjects with a fibre intake in the second (2.6 to 3.1 g per MJ per day), third (3.1 to 3.7 g per MJ per day) and upper quartile (>3.7 g per MJ per day), respectively compared to subjects in the reference quartile (<2.6 g per MJ per day). The lowest risk was observed among subjects with both a high fibre (above median, >3.1 g per MJ per day) and low fat (below median, <33.2 E%) intake.

Several prospective cohort studies have investigated the relationship between dietary fibre intake and risk of type 2 diabetes. Most studies have found a decrease in risk with increasing intakes of cereal fibre (Murakami et al., 2005; Krishnan et al., 2007; Schulze et al., 2007) and whole grains (de Munter et al., 2007). Fewer studies have found a significant risk reduction with total dietary fibre intake (Salmerón et al., 1997a and 1997b; Meyer et al., 2000; Montonen et al., 2003). In the latter studies, total dietary fibre intakes of about 25 to 40 g per day have been consistently associated with a significantly lower risk of developing type 2 diabetes compared to fibre intakes of about 12 to 16 g per day.

In conclusion, dietary fibre intakes reported to be associated with a reduced risk for type 2 diabetes are >2.6 g per MJ or about 25 to 30 g per day, although the contribution of dietary fibre *per se* to this effect remains to be established.

#### 5.3.9. Cardiovascular disease

In a meta-analysis of 10 prospective cohort studies an increase in the energy-adjusted fibre intake of 10 g per day was associated with a 14% lower risk of all coronary events (fatal and non-fatal myocardial infarction (MI)) and with a 27% lower risk of coronary death (Pereira et al., 2004). The results also indicate that a reduced relative risk was seen for subjects with an energy-adjusted fibre intake of >24 g per day compared to subjects in the reference category (18 to <21 g per day). The U.S. Food and Nutrition Board used data from three cohort studies regarding coronary heart disease (CHD) as a basis for setting an AI for total dietary fibre (IoM, 2005). The AI (14 g per 1000 kcal, 3.4g per MJ) was derived from the upper quintiles of energy-adjusted intake of dietary fibre.

In conclusion, there is epidemiological evidence for a protective effect of dietary fibre intake >24g per day on cardiovascular disease risk.

### 5.4. Glycaemic index and glycaemic load

#### 5.4.1. Glucose tolerance and insulin sensitivity

Short- to medium-term (2 weeks to 6 months) intervention studies have shown that diets with reduced GI can improve markers of metabolic control in diabetes type 1 and 2 (Opperman et al., 2004).

A few intervention studies have investigated the role of GI or GL in healthy subjects in relation to the risk of developing IGT or impaired insulin sensitivity, while controlling for dietary fibre intake.

In a 10-week controlled intervention study (Sloth et al., 2004), healthy overweight subjects (BMI: 25 to 30 kg/m$^2$) were given fat-reduced (22 E%) diets with 57 to 58 E% as total carbohydrates *ad libitum* with either high or reduced GI, but with otherwise similar nutrient composition including dietary fibre content. Both groups lost weight. No significant differences between the groups with respect to metabolic markers such as blood glucose, insulin concentrations or insulin resistance (HOMA-IR) were observed. GI differed by 24 units. Similarly, Philippou et al., (2009) did not observe an effect of low (50) vs high (64) GI diets on insulin sensitivity or beta cell function assessed by HOMA-IR and HOMA-β, respectively, while controlling for the amount of dietary fibre intake (11 vs 13 g per day), whereas Frost et al., (1998) showed a significant increase in insulin sensitivity assessed by a short intravenous glucose tolerance test after consumption of a low GI diet (67 to 71) compared to a high GI diet (87 to 89) both comparable for the amount of dietary fibre (19 vs 18g per day).

In other intervention studies investigating the effects of low and high GI diets consumed *ad libitum* on glucose tolerance or insulin sensitivity, dietary fibre intakes are consistently higher in the low GI diet group and do not generally show an effect of low-GI diets on insulin sensitivity (De Rougemont et al., 2007; Wolever and Mehling, 2002; Wolever and Mehling, 2003; Bouche et al., 2002; Brynes et al., 2003). In an intervention study by Clapp and Lopez, (2007), insulin resistance assessed by the HOMA-IR and the quantitative insulin-sensitivity check (QUICKI) indexes significantly decreased after consumption of a low-GI diet (59) compared to a high GI diet (92) in women. Dietary fibre intake was not reported in the study. In an observational study including 979 adults, Liese et al. (2005) found no relation between GI, GL or carbohydrate intake and measures of insulin sensitivity, insulin secretion, and adiposity in adults with normal or impaired glucose tolerance. Data from the same study showed that average fasting glucose, 2 h plasma glucose after an oral glucose tolerance test and glycated haemoglobin (HbA1c) concentrations were not related to either GI or GL calculated from food frequency questionnaires (and GL adjusted for total energy intake), neither at baseline nor at a 5-year follow-up examination (Mayer-Davis et al., 2006). Also Lau et al. (2005) did not find any association between GI or high GL and insulin resistance assessed by HOMA-IR among 5,675 healthy adults aged 30 to 60 years.

In conclusion, results from observational and mainly short-term intervention studies with controlled diets have given conflicting results with respect to the importance of GI for blood glucose control and insulin sensitivity. The data available do not allow setting a DRV for GI/GL based on this outcome.

### 5.4.2. Serum lipids

Intervention studies with controlled diets have given conflicting results with respect to effects of GI/GL on serum lipids. This conflict might be due to methodological problems in designing experimental diets that are similar in dietary composition except for GI. In the well-controlled 10-week intervention study by Sloth et al. (2004) in healthy overweight subjects, who received a fat-reduced diet (21 to 23 E% fat, 57 to 58 E% carbohydrates) with either a high- or low-GI, there were no significant differences between the groups with respect to TG and HDL-cholesterol concentrations. However, a 10% reduction in LDL-cholesterol concentrations was obtained in the group that received the low-GI diet. These findings are supported by a meta-analysis of 15 intervention studies comparing effects of low-GI diets with high-GI diets on serum lipids and other risk factors for coronary heart disease (Kelly et al., 2004).

Some cross-sectional epidemiological studies have indicated that diets with a high GI or GL are associated with unfavourable effects on serum lipids (Augustin et al., 2002). For example Liu et al. (2001) found that fasting plasma TG concentration in 185 healthy postmenopausal women was positively related to GL, especially in overweight and obese subjects. GL was also inversely associated with non-fasting HDL-cholesterol concentration. However, there was no association with the energy adjusted total carbohydrate intake. In a cohort study of 355 healthy adults 35 to 65 years old, Oxlund and Heitmann (2006) found that dietary GI was directly related to changes in total and LDL-cholesterol concentrations in men, but not in women, after six years follow-up. No significant relationships were seen for HDL-cholesterol or TG concentrations. Associations were weak and generally confined to some subgroups, e.g. age categories.

In conclusion, effects on serum cholesterol concentrations are largely dependent on the amount and proportion of fatty acids in the diet, but diets with a low GI may contribute to lowering LDL cholesterol. However, the data available are insufficient to set a DRV for GI/GL based on their effects on serum lipids.

### 5.4.3. Body weight

A systematic review of 6 selected controlled intervention studies including in total 202 overweight or obese subjects showed that weight reduction was about 1 kg greater in subjects allocated to a diet with reduced GI or GL compared to controls (Thomas et al., 2007). Study duration varied between 6 weeks to 6 months. The studies used different designs, e.g. *ad libitum*/energy restriction, low-GI/low-GL or combinations of these factors. Also macronutrient composition varied widely among the studies. The differences in GI between intervention and control diets varied considerably, from 4 to 7 units to 25 to 30 units. In the three studies that used an *ad libitum* design no significant differences in final body weight were found (Bouché et al., 2002; Sloth et al., 2004; Ebbeling et al., 2005). Only one study (Sloth et al. 2004) compared diets with similar nutrient composition, with no significant difference in weight change after 10 weeks. Other short- to medium-term (12 weeks to 4 months) intervention studies in which diets were administered *ad libitum* to mainly overweight and obese subjects have not shown differences in weight change related to GI (Wolever and Mehling, 2002 and 2003; Aston et al., 2008).

Controlled intervention studies of longer duration (1 to 1.5 years) do not show major differences in weight change in overweight/obese subjects related to GI/GL (Ebbeling et al., 2007; Das et al., 2007) or normal/overweight subjects (Sichieri et al., 2007).

Results from cohort studies regarding the relation between GI and/or GL on body weight are equivocal (Gaesser, 2007; McMillan-Price et al., 2006, McMillan-Price and Brand-Miller, 2006; Sloth and Astrup, 2006; Thorsdottir and Birgisdottir, 2005).

In conclusion, available studies do not allow a firm conclusion with respect to effects of diets with different GI and/or GL on body weight, neither to set a DRV for GI/GL based on this outcome. Most studies have been short-term and there are few controlled studies with *ad libitum* food intake. The difference in GI or GL between intervention and control has varied between studies and diets have also differed in other nutritional aspects, e.g. dietary fibre intake and/or energy density. In studies comparing low GL with high GL, macronutrient composition has differed between high and low GL diets, whereas GI may or may not have differed depending on the strategy used for GL manipulation (changes in carbohydrate content, in GI of carbohydrate containing foods, or both).

### 5.4.4. Type 2 diabetes mellitus

Prospective cohort studies comparing diets with different GI or GL have shown conflicting results with respect to the risk of developing type 2 diabetes (Salmerón et al., 1997a; Salmerón et al., 1997b; Meyer et al., 2000; Hu et al., 2001; Stevens et al., 2002; Hodge et al., 2004; Schulze et al., 2004b; Krishnan et al,. 2007; Mosdøl et al., 2007; Sahyoun et al., 2008; Halton et al., 2008). A meta-analysis including eight studies found an increased risk of diabetes when comparing highest to lowest quintiles of both GI and GL (Barclay et al., 2008). However, the studies by Krishnan et al. (2007), Sahyoun et al. (2008) and Mosdøl et al. (2007), which showed no or an inverse relationship, were not included. Adjustment for dietary factors was limited to dietary fibre intake.

In conclusion, few observational studies indicate that diets with either low GI or low GL might be associated with a decreased risk of developing type 2 diabetes, but data are inconsistent and do not allow setting a DRV for GI/GL on the basis of type 2 diabetes risk.

### 5.4.5. Cardiovascular disease

Data from the Nurses Health Study show a positive association of GL with risk of cardiovascular events (MI, CHD deaths, Liu et al., 2000; Halton et al., 2006) or stroke in overweight women (Oh et al. 2005). A study among elderly men did not observe any relation between GI and CVD risk (van Dam et al., 2000). GL was not considered in the study.

In a prospective study of 36,246 Swedish men aged 45 to 79 years without diabetes or prior cardiovascular disease, dietary GI and dietary GL were not associated with ischaemic cardiovascular disease or mortality after 6 years follow-up (Levitan et al., 2007a). However, a weak trend for a greater risk of haemorrhagic stroke with increasing GL was observed. In a subsequent study of 4,617 men aged 45 to 79 years with prior cardiovascular disease, dietary GI and GL were not associated with cardiovascular or all-cause mortality after 6 years follow-up (Levitan et al., 2007b). In a recent systematic review of the evidence supporting a causal link between dietary factors and CHD, high GI and high GL were among those showing the strongest associations with increased risk of CHD (Mente et al., 2009).

In conclusion, a few observational studies indicate that diets with high GL might be associated with an increased risk of CVD, but data are inconclusive and do not allow setting a DRV for GI/GL on the basis of CVD risk.

### 5.4.6. Colorectal cancer

A meta-analysis of 4 case-control and 7 prospective cohort studies found a significantly increased pooled risk of colorectal cancer for the upper compared to the lower quintile of GL (Gnagnarella et al., 2008). There was, however, large heterogeneity between studies and risk estimates were not significant for prospective studies.

In conclusion, the data available do not allow setting a DRV for GI/GL on the basis of colorectal cancer risk.

## 6. Data on which to base dietary reference values

### 6.1. Total and glycaemic carbohydrates

Reference values for the intake of glycaemic carbohydrate have to take into account the amount of energy to be provided when reference intakes for protein and fat intake have been met.

The absolute dietary requirement for glycaemic carbohydrates is not precisely known but will depend on the amount of fat and protein ingested. Generally an intake of 50 to 100 g per day will prevent ketosis. An intake of 130 g per day for both children (>1 years) and adults has been estimated to be sufficient to cover the needs of glucose for the brain. This intake corresponds to about 18 and 25 E% in adult males and females assuming an energy intake of 2,800 and 2,100 kcal per day (11,700 and 8,780 kJ per day), respectively. However, these levels of intake are not sufficient to meet energy needs in the context of acceptable intake levels of fat and protein.

Intervention studies provide evidence that high fat, low carbohydrate diets consumed *ad libitum* are associated with an increase in body weight, but data are insufficient to define an LTI for carbohydrates. High carbohydrate diets tend to induce adverse effects on the lipid profile, but there is an insufficient scientific basis for setting an UL for total carbohydrates. The Panel therefore comes to the conclusion that only a Reference Intake Range can be given for total carbohydrate intake, partly based on practical considerations (e.g. current levels of intake, achievable dietary patterns) (see section 3).

Based on the above considerations the Panel proposes 45 to 60 E% as the Reference Intake Range for carbohydrates. Diets with glycaemic carbohydrate contents of 45 to 60 E%, in combination with reduced intakes of fat and SFA, are compatible with the improvement of metabolic risk factors for chronic disease,

as well as with mean carbohydrate intakes observed in some European countries. This intake range applies to both adults and children older than one year of age.

### 6.2. Sugars

Frequent consumption of sugar-containing foods can increase risk of dental caries, especially when prophylactic measures, e.g. oral hygiene and fluoride prophylaxis, are insufficient. However, available data do not allow the setting of an UL for (added) sugars on the basis of a risk reduction for dental caries, as caries development related to consumption of sucrose and other cariogenic carbohydrates does not depend only on the amount of sugar consumed, but it is also influenced by oral hygiene, exposure to fluoride, frequency of consumption, and various other factors.

The evidence relating high intake of sugars (mainly as added sugars), compared to high intakes of starch, to weight gain is inconsistent for solid foods. However, there is some evidence that high intakes of sugars in the form of sugar-sweetened beverages might contribute to weight gain. The available evidence is insufficient to set an upper limit for sugars based on their effects on body weight.

Observed negative associations between added sugar intake and micronutrient density of the diet are mainly related to patterns of intake of the foods from which added sugars in the diet are derived rather than to the intake of added sugars *per se*. The available data are not sufficient to set an upper limit for (added) sugar intake.

Most short-term intervention studies on the effects of high intakes of sugars on glucose and insulin response do not find adverse effects at intakes of predominantly added sugars up to 20 to 25 E%, provided that body weight is maintained. Although there is some evidence that high intakes (>20 E%) of sugars may increase serum TG and cholesterol concentrations, and that intakes >20-25 E% might adversely affect glucose and insulin response, the available data are not sufficient to set an upper limit for (added) sugar intake.

Evidence on the relationship of patterns of consumption of sugar-containing foods to dental caries, weight gain and micronutrient intake should be considered when establishing nutrient goals for populations and recommendations for individuals and when developing food-based dietary guidelines.

The Panel notes that some authorities have established upper limits for population average intake or individual intake of added sugars of <10 E% but others have not (see Section 4). Typically, these nutrient recommendations reflect a judgement of what level of sugar intake is practically achievable within the context of a nutritionally adequate diet based on known patterns of intake of foods and nutrients in specific populations. It is also noted that the average intake of (added) sugars in some EU Member States exceed 10 E%, especially in children.

### 6.3. Dietary fibre

The role of dietary fibre in bowel function was considered the most suitable criterion for establishing an adequate intake. Based on the available evidence on bowel function, the Panel considers dietary fibre intakes of about 25 g per day to be adequate for normal laxation in adults.

The Panel notes that there is evidence in adults of benefit to health associated with consumption of diets rich in fibre-containing foods at dietary fibre intakes greater than 25 g per day, e.g. reduced risk of coronary heart disease and type 2 diabetes and weight maintenance.

The Panel considers that the AI for dietary fibre for children should be based on that for adults (25 g, equivalent to 2 to 3 g per MJ for daily energy intakes of 8 to 12 MJ) with appropriate adjustment for energy intake. 2 g per MJ is considered to be adequate for normal laxation in children. Table 3 shows AI for fibre in children of different ages based on average energy intakes. Available evidence indicates that a fibre intake corresponding to 2 to 2.5 g per MJ is compatible with normal growth and development in children.

### 6.4. Glycaemic index and glycaemic load

Although there is some support for a role of GI and GL in the treatment of type-2 diabetes and some evidence suggesting that lowering GI and GL may have favourable effects on some metabolic risk factors such as serum lipids, the evidence regarding their role in the prevention of diet-related diseases is still inconclusive.

## CONCLUSIONS

### Total and glycaemic carbohydrates

The Panel considers that there is insufficient scientific basis for setting a Lower Threshold Intake (LTI), a Population Reference Intake (PRI), an Adequate Intake (AI) or a Tolerable Upper Intake Level (UL) for total carbohydrates.

Based on the effects of carbohydrates (and fat) intakes on body weight and blood lipids, while taking into account practical considerations (e.g. current levels of intake, achievable dietary patterns), the Panel proposes 45 to 60 E% as the Reference Intake range for carbohydrates. This intake range applies to both adults and children from one year of age.

### Sugars

Available data do not allow the setting of a Tolerable Upper Intake Level for total or added sugars, neither an Adequate Intake nor a Reference Intake range.

### Dietary fibre

The role of dietary fibre in bowel function was considered the most suitable criterion for establishing an adequate intake. Based on the available evidence on bowel function, the Panel considers dietary fibre intakes of 25 g per day to be adequate for normal laxation in adults. There is limited evidence to set adequate intakes for children. The Panel considers that the AI for dietary fibre for children should be based on that for adults with appropriate adjustment for energy intake. A fibre intake of 2 g per MJ is considered adequate for normal laxation in children from the age of one year.

The Panel notes that there is evidence in adults of benefit to health associated with consumption of diets rich in fibre-containing foods at dietary fibre intakes greater than 25 g per day, e.g reduced risk of coronary heart disease and type 2 diabetes and improved weight maintenance. Such evidence should be considered when developing food-based dietary guidelines.

### Glycaemic index and glycaemic load

The evidence regarding the role of the “gylcaemic index” and the “glycaemic load” in prevention of diet-related diseases is still inconclusive.

**Table 3.** Summary of Dietary Reference Values for carbohydtares and dietary fibre.

| Category | Adults | Children |
|---|---|---|
| Total carbohydrates, E% (RI) | 45-60 | 45 to 60 (from 1 year of age) |
| Dietary Fibre, g/day (AI) | 25 | 10 (from 1 to 3 years)<br>14 (from 4 to 6 years)<br>16 (from 7 to 10 years)<br>19 (from 11 to 14 years)<br>21 (from 15 to 17 years) |

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# ANNEXES

## ANNEX 1 DEFINITIONS OF DIETARY FIBRE IN RECOMMENDATIONS OF FIBRE INTAKE

| Source | Definition |
|---|---|
| Commission Directive 2008/100/EC[9] | For the purposes of this Directive "fibre" means carbohydrate polymers with three or more monomeric units, which are neither digested nor absorbed in the human small intestine and belong to the following categories:<br>- edible carbohydrate polymers naturally occurring in the food as consumed;<br>- edible carbohydrate polymers which have been obtained from food raw material by physical, enzymatic or chemical means and which have a beneficial physiological effect demonstrated by generally accepted scientific evidence;<br>- edible synthetic carbohydrate polymers which have a beneficial physiological effect demonstrated by generally accepted scientific evidence." |
| SACN, 2008 | Material that is resistant to digestion and absorption in the small intestine and that has a demonstrable physiological effect potentially associated with health benefits in the body, such as increasing stool bulk, decreasing intestinal transit time or decreasing post prandial glycaemia. This includes NSP and soluble fibre. Inclusion of other components in the definition would require physiological effects to be demonstrated. |
| FAO/WHO (Mann et al., 2007) | Intrinsic plant cell wall polysaccharides, NSP. Dietary fibre should reflect the health benefits of a diet rich in fruits, vegetables and whole grains. |
| GR, 2006 | Carbohydrates, compounds analogous to carbohydrates, and lignin and related substances that are not digested or absorbed in the human small intestine. These include:<br>Polysaccharides other than starch, and indigestible oligosaccharides: e.g. cellulose, hemicelluloses such as arabinoxylans, arabinogalactans and xyloglucans, pectin, fructans and some oligosaccharides (inulin, fructo-oligosaccharides, oligofructose), galacto-oligosaccharides and xylo-oligosaccharides, gums and mucilages (for some population groups: lactose)<br>Compounds analogous to carbohydrates: indigestible dextrins (mainly from potatoes and maize), synthetic carbohydrates and their derivatives, polydextrose, methylcellulose, hydroxypropyl methylcellulose, etc. ndigestible starch.<br>Lignin.<br>Substances that occur in products containing lignin or polysaccharides other than starch: wax, cutin, saponins, suberins, tannins. |
| IoM 2005 | Dietary fibre: non-digestible carbohydrates and lignin that are intrinsic and intact in plants, e.g. cellulose, pectin, gums, hemicellulose, b-glucans, and fibres contained in oat and wheat bran, plant carbohydrates that are not recovered by alcohol precipitation (e.g. inulin, oligosaccharides, and fructans), lignin, and some resistant starch. Excluded are non-digestible mono- and disaccharides and polyols, some resistant starch, non-digestible animal carbohydrates.<br>Functional fibre: isolated, non-digestible carbohydrate components that have beneficial physiological effects in humans. May be isolated or extracted using chemical, enzymatic, or aqueous steps. Synthetically manufactured (DP $\geq$3) or naturally occurring isolated oligosaccharides and manufactured resistant starch are included. Naturally occurring polysaccharides or oligosaccharides usually extracted from their plant source that have been modified (e.g. to a shorter polymer length or to a different molecular arrangement) and animal derived non-digestible carbohydrates are included. Excluded are non-digestible mono- |

[9] Commission Directive 2008/100/EC of 28 October 2008 amending Council Directive 90/496/EEC on nutrition labelling for foodstuffs as regards recommended daily allowances, energy conversion factors and definitions. OJ L 285, 29.10.2008, pp. 9-12

| Source | Definition |
|---|---|
| | and disaccharides and polyols, some resistant starch, non-digestible animal carbohydrates.<br>Total dietary fibre: sum of Dietary and Functional fibre. |
| AFSSA, 2001 | Dietary constituents not digested by the enzymes of the gastrointestinal tract, mainly plant-derived constituents, e.g. cellulose, hemicellulose, lignin, gums, alginates, carragenans, resistant starch. Minor dietary components such as oligosaccharides (fructo-oligosaccharides, a-galactosides, lactulose), constituents produced by micro-organisms (xanthanes), constituents of crustaceans (e.g. chitine, chitosane) are also included. |
| D-A-CH, 2008 | Dietary fibre comprises those components of vegetable food which are not degraded by physiological enzymes of the human gastrointestinal tract. Dietary fibre, except for lignin, stands for indigestible carbohydrates such as cellulose, hemicellulose, pectin etc. Resistant starch and indigestible oligosaccharides such as oligofructose and oligosaccharides of the raffinose family (raffinose, stachyose, verbascose in pulses) are included. |
| NNR, 2004 | Dietary fibre recommendation refers to dietary fibre naturally occurring in plant foods as measured by AOAC methods for total dietary fibre. |
| Codex proposal, Alinorm 09/32/26, 2009 | Dietary fibre means carbohydrate polymers[1] with ten or more monomeric units[2], which are not hydrolysed by the endogenous enzymes in the small intestine of humans and belong to the following categories: :<br>Edible carbohydrate polymers naturally occurring in the food as consumed,<br>carbohydrate polymers, which have been obtained from food raw material by physical, enzymatic or chemical means and which have been shown to have a physiological effect of benefit to health as demonstrated by generally accepted scientific evidence to competent authorities,<br>synthetic carbohydrate polymers which have been shown to have a physiological effect of benefit to health as demonstrated by generally accepted scientific evidence to competent authorities<br>Properties:<br>Dietary fibre generally has properties such as:<br>• Decrease intestinal transit time and increase stools bulk<br>• fermentable by colonic microflora<br>• Reduce blood total and/or LDL cholesterol levels<br>• Reduce post-prandial blood glucose and /or insulin levels.<br>[1] When derived from a plant origin, dietary fibre may include fractions of lignin and/or other compounds when associated with polysaccharides in the plant cell walls and if these compounds are quantified by the AOAC gravimetric analytical method for dietary fibre analysis : Fractions of lignin and the other compounds (proteic fractions, phenolic compounds, waxes, saponins, phytates, cutin, phytosterols, etc.) intimately "associated" with plant polysaccharides are often extracted with the polysaccharides in the AOAC 991.43 method. These substances are included in the definition of fibre insofar as they are actually associated with the poly- or oligo-saccharidic fraction of fibre. However, when extracted or even re-introduced into a food containing non digestible polysaccharides, they cannot be defined as dietary fibre. When combined with polysaccharides, these associated substances may provide additional beneficial effects (pending adoption of Section on Methods of Analysis and Sampling).<br>[2] Decision on whether to include carbohydrates from 3 to 9 monomeric units should be left to national authorities. |
| DoH,1991 | NSP (non alpha-glucan polysaccharides): cellulose, non-cellulose polysaccharides (pectins, glucans, arabinogalactans, arabinoxylans, gums, mucilages, inulin, guar, chitin. |

## ANNEX 2A POPULATION, METHODS AND PERIOD OF DIETARY ASSESSMENT IN CHILDREN AND ADOLESCENTS IN EUROPEAN COUNTRIES

| Country | Population | Dietary method | Year of survey | Reference |
|---|---|---|---|---|
| **AT** | Boys and girls aged 7-9 years | 3-day record | 2007 | Elmadfa et al., 2009 |
| | Boys and girls aged 10-14 years | 3-day record | 2007 | Elmadfa et al., 2009 |
| | Boys and girls aged 14-19 years | 24-hour recall | 2003-2004 | Elmadfa et al., 2009 |
| **BE** | Boys and girls aged 2.5-3 years | 3-day record | 2002-2003 | Huybrechts and DeHenauw, 2007 |
| | Boys and girls aged 4-6.5 years | 3-day record | 2002-2003 | Huybrechts and DeHenauw, 2007 |
| | Boys and girls aged 13-15 years | 7-day record | 1997 | Matthys et al., 2003 |
| | Boys and girls aged 15-18 | 2x 24-hour recall | 2004 | De Vriese et al., 2006 |
| **CZ** | Boys and girls aged 4-6 years | 2x 24-hour recall | n.a.[1] | Tláskas, Hrstková.(unpublished data) (In: Elmadfa, 2009) |
| | Boys and girls aged 7-9 years | 2x 24-hour recall | n.a. | Tláskas, Hrstková. (unpublished data) (In:Elmadfa, 2009) |
| **DE** | Infants aged 12 months | 3-day record | 1989-2003 | Hilbig and Kersting, 2006 |
| | Children aged 18 months | 3-day record | 1989-2003 | Hilbig and Kersting, 2006 |
| | Children aged 2 years | 3-day record | 1989-2003 | Hilbig and Kersting, 2006 |
| | Children aged 3 years | 3-day record | 1989-2003 | Hilbig and Kersting, 2006 |
| | Boys and girls aged 6 years | 3-day record | 2006 | Mensink et al., 2007 |
| | Boys and girls aged 7-9 years | 3-day record | 2006 | Mensink et al., 2007 |
| | Boys and girls aged 10-11 years | 3-day record | 2006 | Mensink et al., 2007 |
| | Boys and girls aged 12 years | Dietary history (over the last 4 weeks) | 2006 | Mensink et al.,2007 |
| | Boys and girls aged 13-14 years | Dietary history (over the last 4 weeks) | 2006 | Mensink et al.,2007 |
| | Boys and girls aged 15-17 years | Dietary history (over the last 4 weeks) | 2006 | Mensink et al.,2007 |
| **DK** | Boys and girls aged 1-3 years | 7-day record | 1995 | Andersen et al., 1996 |
| | Boys and girls aged 4-5 years | 7-day record | 2000-2002 | Lyhne et al., 2005 |
| | Boys and girls aged 6-9 years | 7-day record | 2000-2002 | Lyhne et al., 2005 |
| | Boys and girls aged 10-13 years | 7-day record | 2000-2002 | Lyhne et al., 2005 |
| | Boys and girls aged 14-17 years | 7-day record | 2000-2002 | Lyhne et al., 2005 |
| **FI** | Infants aged 8 months | 3-day record | 1999 | Lagstrom, 1999 |
| | Children aged 3 years | 4-day record | 1999 | Lagstrom, 1999 |
| | Children aged 4 years | 4 day record | 1999 | Lagstrom, 1999 |
| | Children aged 4 years | 3-day record | 2008 | Kyttälä et al., 2008 |
| | Children aged 6 years | 3-day record | 2008 | Kyttälä et al., 2008 |
| **FR** | Boys and girls aged 4-6 years | 3x 24-hour recall | 2006-2007 | Castetbon et al. 2009 (In: Elmadfa, 2009) |

| | | | | |
|---|---|---|---|---|
| | Boys and girls aged 7-9 years | 3x 24-hour recall | 2006-2007 | Castetbon et al. 2009 (In: Elmadfa, 2009) |
| | Boys and girls aged 10-14 years | 3x 24-hour recall | 2006-2007 | Castetbon et al. 2009 (In: Elmadfa, 2009) |
| | Boys and girls aged 15-18 years | 3x 24-hour recall | 2006-2007 | Castetbon et al. 2009 (In: Elmadfa, 2009) |
| **GR** | Boys and girls aged 4-5 years | 3-day record+24-hour recall / 3-day record | 2003-2004 | Manios et al., 2008 |
| **HU** | Boys and girls aged 11-14 years | 3x 24-hour recall | 2005-2006 | Biro et al.2007 (In: Elmadfa, 2009) |
| **IE** | Boys and girls 5-8 years | 7-day record | 2003-2004 | Irish Universities Nutrition Alliance, National Irish Children‟s Food Survey. www.iuna.net |
| | Boys and girls 9-12 years | 7-day record | 2003-2004 | Irish Universities Nutrition, Alliance National Irish Children‟s Food Survey. www.iuna.net |
| **IT** | Boys and girls 4-6 years | 7-day record | n.a | D‟Amicis, 2000 |
| | Boys and girls 7-9 years | 7-day record | n.a | D‟Amicis, 2000 |
| | Boys and girls 10-14 years | 7-day record | n.a | D‟Amicis, 2000 |
| | Boys and girls 15-18 years | 7-day record | n.a | D‟Amicis, 2000 |
| **NL** | Infants aged 9 month | 2-day record (independent days) | 2002 | Boer et al., 2006 |
| | Infants aged 12 monts | 2-day record (independent days) | 2002 | Boer et al., 2006 |
| | Children aged 18 months | 2-day record (independent days) | 2002 | Boer et al., 2006 |
| | Boys and girls aged 2-3 years | 2-day record (independent days) | 2005-2006 | Ocke et al., 2008 |
| | Boys and girls aged 4-6 years | 2-day record (independent days) | 2005-2006 | Ocke et al., 2008 |
| | Boys and girls aged 7-9 years | 2-day record | 1997-1998 | Hulshof et al., 1998 |
| | Boys and girls aged 10-12 years | 2-day record | 1997-1998 | Hulshof et al., 1998 |
| | Boys and girls aged 13-15 years | 2-day record | 1997-1998 | Hulshof et al., 1998 |
| | Boys and girls aged 16-19 years | 2-day record | 1997-1998 | Hulshof et al., 1998 |
| **NO** | Children aged 2 years | Food Frequency Questionnaire | 1998-1999 | Lande and Andersen, 2005 |
| | Boys and girls aged 4 years | 4-day record | 2000 | Øverby and Andersen, 2002 |
| | Boys and girls aged 9 years | 4-day record | 2000 | Øverby and Andersen, 2002 |
| | Boys and girls aged 13 | 4-day record | 2000 | Øverby and Andersen, 2002 |
| | Boys and girls aged 16-19 years | Food Frequency Questionnaire | 1997 | Johansson and Solvoll, 1999 |
| **PL** | Boys and girls aged 4-6 years | 24-hour recall | 2000 | Szponar et al, 2000 (unpublished data) (In: Elmadfa, 2009) |
| | Boys and girls aged 7-9 years | 24-hour recall | 2000 | Szponar et al, 2000 (unpublished data) (In: Elmadfa, 2009) |
| | Boys and girls aged 10-14 years | 24-hour recall | 2000 | Szponar et al, 2000 (unpublished data) (In: Elmadfa, 2009) |
| | Boys and girls aged 15-18 years | 24-hour recall | 2000 | Szponar et al, 2000 (unpublished data) (In: Elmadfa, 2009) |
| **PT** | Boys and girls aged 7-9 years | 24-hour recall | 2000-2002 | Moreira et al., 2005 |
| | Boys and girls aged 13 years | 24-hour recall | 2000-2002 | Moreira et al., 2005 |

| | | | | |
|---|---|---|---|---|
| **SI** | Boys and girls aged 14-17 years | Food Frequency Questionnaire | n.a. | Fidler Mis et al. (unpublished data) (In: Elmadfa, 2009) |
| **ES** | Boys and girls aged 10-14 years | 2x 24-hour recall | 2002-2003 | Serra Majem and Ribas, 2007; Serra Majem et al., 2006 and 2007 (In: Elmadfa, 2009) |
| | Boys and girls aged 15-18 years | 2x 24-hour recall | 2002-2003 | Serra Majem and Ribas, 2007; Serra Majem et al., 2006 and 2007 (In: Elmadfa, 2009) |
| **SE** | Boys and girls aged 4 years | 4-day record | 2003 | Enghardt-Barbieri et al., 2006 |
| | Boys and girls aged 8-9 years | 4-day record | 2003 | Enghardt-Barbieri et al., 2006 |
| | Boys and girls aged 11-12 years | 4-day record | 2003 | Enghardt-Barbieri et al., 2006 |
| **UK** | Boys and girls aged 4-6 years | 7-day record | 1997 | Gregory et al., 2000 |
| | Boys and girls aged 7-10 years | 7-day record | 1997 | Gregory et al., 2000 |
| | Boys and girls aged 11-14 years | 7-day record | 1997 | Gregory et al., 2000 |
| | Boys and girls aged 15-18 years | 7-day record | 1997 | Gregory et al., 2000 |

[1]n.a. = not available

## ANNEX 2B INTAKE OF CARBOHYDRATES AND DIETARY FIBRE AMONG CHILDREN AGED ~1-3 YEARS IN EU COUNTRIES.

| Country | Age yrs | N | Total Carbohydrates (E%) | | | Mono- and Disaccharides (E%) | | | Polysaccharides (E%) | | | Sucrose (E%) | | | Dietary Fibre (g) | | | Dietary Fibre (g/MJ) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 |
| **Infants and Toddlers** | | | | | | | | | | | | | | | | | | | | |
| **DE** | 12 mo | 432 | 52.2 | 6.0 | | | | | | | | | | | | | | | | |
| | 18 mo | 478 | 49.8 | 6.8 | | | | | | | | | | | | | | | | |
| | 2 | 458 | 49.4 | 6.6 | | | | | | | | | | | | | | | | |
| | 3 | 427 | 50.3 | 6.3 | | | | | | | | | | | | | | | | |
| **FI** | 8 mo | 215 | 58.0 | 11.0 | | | | | | | | 3.0 | 2.0 | | 6.7 | 2.9 | | | | |
| | 13 mo | 449 | 54.0 | 10.0 | | | | | | | | 5.0 | 3.0 | | 9.0 | 2.9 | | | | |
| | 2 | 398 | 50.0 | 11.0 | | | | | | | | 10.0 | 4.0 | | 9.7 | 2.8 | | | | |
| | 3 | 359 | 51.0 | 10.0 | | | | | | | | 11.0 | 5.0 | | 10.9 | 3.2 | | | | |
| **NL** | 9 mo | 333 | 58.0 | 4.2 | 52.6-63.3 | 36.3 | 5.5 | 29.5-43.4 | | | | | | | | | | 2.4 | 0.7 | 1.6-3.2 |
| | 12 mo | 306 | 57.4 | 4.1 | 52.1-62,7 | 35.7 | 4.9 | 28.6-41.0 | | | | | | | | | | 2.5 | 0.5 | 1.8-3.2 |
| | 18 mo | 302 | 57.5 | 3.9 | 52.5-62.6 | 36.3 | 5.2 | 29.8-43.0 | | | | | | | | | | 2.5 | 0.5 | 2.9-3.2 |
| **NO** | 2 | 172 | 53.3 | 5.6 | | | | | | | | 11.7 | 5.8 | | 13.6 | 5.2 | | | | |
| **Pre-school children** | | | | | | | | | | | | | | | | | | | | |
| **Males** | | | | | | | | | | | | | | | | | | | | |
| **BE** | 2.5-3 | 102 | 54.2 | 5.2 | | 31.6 | 5.2 | | 22.6 | 3.4 | | | | | 14.6 | 3.4 | | | | |
| **DK** | 1-3 | 129 | 51.0 | | | | | | | | | 11.0 | | | 15.0 | | | | | |
| **NL** | 2-3 | 313 | 58.0 | | 49-66 | | | | | | | | | | 13.0 | | 8-19 | 2.3 | | 1.5-3.2 |
| **Females** | | | | | | | | | | | | | | | | | | | | |
| **BE** | 2.5-3 | 95 | 52.9 | 5.4 | | 29.7 | 5.4 | | 23.0 | 4.1 | | | | | 13.0 | 2.8 | | | | |
| **DK** | 1-3 | 149 | 50.0 | | | | | | | | | 11.0 | | | 14.0 | | | | | |
| **NL** | 2-3 | 313 | 58.0 | | 51-65 | | | | | | | | | | 12.0 | | 8-17 | 2.3 | | 1.5-3.1 |

[1]SE; [2] P2.5-P97.5

## ANNEX 2B INTAKE OF CARBOHYDRATES AND DIETARY FIBRE AMONG CHILDREN AGED ~4-6 YEARS IN EU COUNTRIES.

| Country | Age yrs | N | Total Carbohydrates (E%) | | | Mono- and Disaccharides (E%) | | | Polysaccharides (E%) | | | Sucrose (E%) | | | Dietary Fibre (g) | | | Dietary Fibre (g/MJ) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 |
| **Males** | | | | | | | | | | | | | | | | | | | | |
| **BE** | 4-6.5 | 236 | 54.2 | 4.5 | | 31.4 | 5.2 | | 22.7 | 3.3 | | | | | 14.6 | 3.3 | | | | |
| **CZ** | 4-6 | 641 | 56.0 | 5.8 | | | | | | | | | | | | | | | | |
| **DE** | 6 | 106 | 53.3 | 6.2 | 41.8-63.6 | | | | | | | | | | 15.7 | 4.1 | 9.9-24.1 | | | |
| **DK** | 4-5 | 82 | 50.0 | 4.1 | 43.0-56.0 | | | | | | | 12.0 | 4.1 | 6.0-20.0 | 17 | 4.6 | 10.0-24.0 | 2.2 | 0.5 | 1.5-3.1 |
| **FI** | 4 | 307 | 53.0 | | | | | | | | | 13.7 | | | 9.6 | 3.2 | | | | |
| | 6 | 364 | 53.0 | | | | | | | | | 13.0 | | | 11.4 | 3.7 | | | | |
| **FR** | 4-6 | 164 | 49.8 | 0.8[1] | | | | | | | | | | | 11.8 | 0.5[1] | | | | |
| **GR** | 4-5 | 356 | 44.8 | 6.5 | | | | | | | | | | | | | | | | |
| **IT** | 4-6 | 21 | 50.0 | 5.5 | | | | | | | | | | | 14.9 | 4.7 | | | | |
| **NL** | 4-6 | 327 | 56.8 | 0.9 | 48.0-64.0 | | | | | | | 13.0 | 2.0 | | 14 | 3 | 9.0-20.0 | 2.2 | | 1.5-3.0 |
| **NO** | 4 | 206 | 53.0 | 5.0 | | | | | | | | 15.0 | 5.0 | | 12.0 | 5.0 | | | | |
| **PL** | 4-6 | 82 | 57.0 | 7.9 | | | | | | | | 18.5 | 5.8 | | 16.8 | 5.8 | | | | |
| **SE** | 4 | 302 | 54.2 | 4.8 | 46.5-62.4 | 28.6 | | | | | | 13.8 | 4.6 | 7.0-21.6 | 12.0 | 3.0 | 7.0-17.0 | 1.8 | 0.5 | 1.1-2.7 |
| **UK** | 4-6 | 184 | 51.6 | 4.3 | 43.0-59.4 | | | | | | | | | | | | | | | |
| **Females** | | | | | | | | | | | | | | | | | | | | |
| **BE** | 4-6.5 | 228 | 54.9 | 4.1 | | 31.3 | 5.1 | | 23.4 | 3.4 | | | | | 13.9 | 3.2 | | | | |
| **CZ** | 4-6 | 446 | 56.0 | 5.8 | | | | | | | | | | | 15.3 | 4.7 | | | | |
| **DE** | 6 | 102 | 53.3 | 5.2 | 41.8-63.6 | | | | | | | | | | 15.8 | 4.7 | 8.6-24.7 | | | |
| **DK** | 4-5 | 116 | 50.0 | 4.1 | 43.0-56.0 | | | | | | | 12.0 | 3.9 | 7.0-20.0 | 16 | 4.5 | 9.0-22.0 | 2.2 | 0.5 | 1.5-3.0 |
| **FI** | 4 | 307 | 53.0 | | | | | | | | | 13.6 | | | 9.4 | 3.0 | | | | |
| | 6 | 349 | 53.0 | | | | | | | | | 13.8 | | | 10.3 | 3.3 | | | | |
| **FR** | 4-6 | 162 | 48.6 | 0.5[1] | | | | | | | | | | | 11.5 | 0.3[1] | | | | |
| **GR** | 4-5 | 389 | 45.2 | 6.4 | | | | | | | | | | | | | | | | |
| **IT** | 4-6 | 17 | 50.3 | 4.9 | | | | | | | | | | | 15.8 | 3.9 | | | | |
| **NL** | 4-6 | 312 | 57.0 | 4.0 | 50.0-64.0 | | | | | | | | | | 13 | 3 | 8.0-17.0 | 2.0 | | 1.4-2.8 |
| **NO** | 4 | 185 | 54.0 | 6.0 | | | | | | | | 16.0 | 6.0 | | 12.0 | 6.0 | | | | |
| **PL** | 4-6 | 84 | 55.6 | 7.5 | | | | | | | | 17.9 | 6.0 | | 14.6 | 5.9 | | | | |
| **SE** | 4 | 288 | 53.4 | 5.1 | 45.4-61.6 | 28.4 | | | | | | 13.7 | 4.5 | 6.9-21.2 | 11.0 | 3.0 | 7.0-17.0 | 1.8 | 0.4 | 1.2-2.6 |
| **UK** | 4-6 | 171 | 51.4 | 5.0 | 42.1-60.5[2] | | | | | | | | | | | | | | | |

[1]SE; [2] P2.5-P97.5

## ANNEX 2B INTAKE OF CARBOHYDRATES AND DIETARY FIBRE AMONG CHILDREN AGED ~7-9 YEARS IN EU COUNTRIES.

| Country | Age yrs | N | Total Carbohydrates (E%) | | | Mono- and Disaccharides (E%) | | | Polysaccharides (E%) | | | Sucrose (E%) | | | Dietary Fibre (g) | | | Dietary Fibre (g/MJ) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 |
| **Males** | | | | | | | | | | | | | | | | | | | | |
| **AT** | 7-9 | 146 | 51.4 | 6.8 | | | | | | | | 16.6 | 7.1 | | 15.0 | 5.9 | | | | |
| **CZ** | 7-9 | 940 | 53.4 | 6.7 | | | | | | | | | | | | | | | | |
| **DE** | 7-9 | 321 | 53.2 | 6.1 | 43.6-63.1 | | | | | | | | | | 17.5 | 5.3 | 10.6-26.2 | | | |
| **DK** | 6-9 | 174 | 51.0 | 3.9 | 44.0-57.0 | | | | | | | 13.0 | 5.2 | 6.0-22.0 | 18.0 | 5.8 | 11.0-28.0 | 2.0 | 0.5 | 1.3-2.9 |
| **FR** | 7-9 | 160 | 49.3 | 0.5[1] | | | | | | | | | | | 13.5 | 0.4[1] | | | | |
| **IE** | 5-8 | 145 | 52.4 | 4.8 | 44.5-60.2 | | | | | | | | | | | | | | | |
| **IT** | 7-9 | 29 | | | | | | | | | | | | | 18.5 | | | | | |
| **NL** | 7-9 | 104 | 52.7 | 6.6 | 38.0-63.6 | 29.6 | 7.0 | 18.0-40.5 | 23.0 | 4.0 | 17.0-30.0 | | | | 17.0 | 6.0 | 9.0-25.0 | 2.0 | 0.6 | 1.2-3.0 |
| **NO** | 9 | 402 | 54.0 | 6.0 | | | | | | | | 16.0 | 6.0 | | 16.0 | 7.0 | | | | |
| **PL** | 7-9 | 101 | 56.3 | 7.9 | | | | | | | | 17.3 | 5.6 | | 19.6 | 6.8 | | | | |
| **PT** | 7-9 | 1541 | 48.6 | 7.8 | | | | | | | | 22.5 | 7.1 | | 20.2 | 8.1 | | | | |
| **SE** | 8-9 | 444 | 53.0 | 4.8 | 45.3-60.5 | 25.7 | | | | | | 12.5 | 4.3 | 5.7-20.2 | 14.0 | 4.0 | 8.0-22.0 | 1.7 | 0.4 | 1.2-2.4 |
| **UK** | 7-10 | 256 | 52.4 | 4.1 | 44.2-60.5 | | | | | | | | | | | | | | | |
| **Females** | | | | | | | | | | | | | | | | | | | | |
| **AT** | 7-9 | 134 | 52.2 | 7.0 | | | | | | | | 18.0 | 6.9 | | 14.3 | 4.4 | | | | |
| **CZ** | 7-9 | 765 | 53.4 | 6.7 | | | | | | | | | | | | | | | | |
| **DE** | 7-9 | 308 | 54.2 | 6.7 | 43.1-66.1 | | | | | | | | | | 16.8 | 5.4 | 10.0-26.2 | | | |
| **DK** | 6-9 | 157 | 51.0 | 4.3 | | | | | | | | 13.0 | 4.7 | 7.0-21.0 | 17.0 | 4.5 | 10.0-25.0 | 2.1 | 0.5 | 1.4-2.9 |
| **FR** | 7-9 | 14 | 48.5 | 0.7[1] | | | | | | | | | | | 12.2 | 0.4[1] | | | | |
| **IE** | 5-8 | 151 | 51.5 | 4.6 | 43.4-59.6 | | | | | | | | | | | | | | | |
| **IT** | 7-9 | 21 | | | | | | | | | | | | | 15.2 | 5.0 | | | | |
| **NL** | 7-9 | 134 | 52.0 | 7.3 | 39.6-63.7 | 29.5 | 7.2 | 18.4-41.5 | 22.4 | 4.3 | 15.7-30.2 | | | | 15.0 | 5.0 | 7.0-23.0 | 1.9 | 0.5 | 1.2-2.8 |
| **NO** | 9 | 408 | 55.0 | 6.0 | | | | | | | | 18.0 | 6.0 | | 14.0 | 6.0 | | | | |
| **PL** | 7-9 | 103 | 55.5 | 7.7 | | | | | | | | 16.4 | 6.2 | | 17.4 | 6.7 | | | | |
| **PT** | 7-9 | 1503 | 48.3 | 7.9 | | | | | | | | 21.8 | 7.1 | | 19.4 | 8.2 | | | | |
| **SE** | 8-9 | 445 | 53.3 | 4.9 | 44.7-61.1 | 25.5 | | | | | | 12.6 | 4.2 | 6.1-19.4 | 13.0 | 4.0 | 8.0-19.0 | 1.8 | 0.4 | 1.2-2.6 |
| **UK** | 7-10 | 226 | 51.3 | 4.3 | 42.6-59.9[2] | | | | | | | | | | | | | | | |

[1]SE; [2] P2.5-97.5

## ANNEX 2B INTAKE OF CARBOHYDRATES AND DIETARY FIBRE AMONG CHILDREN AGED ~10-14 YEARS IN EU COUNTRIES.

| Country | Age yrs | N | Total Carbohydrates (E%) | | | Mono- and Disaccharides (E%) | | | Polysaccharides (E%) | | | Sucrose (E%) | | | Dietary Fibre (g) | | | Dietary Fibre (g/MJ) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 |
| **Males** | | | | | | | | | | | | | | | | | | | | |
| **AT** | 10-14 | 248 | 50.8 | 7.5 | | | | | | | | 17.6 | 8.2 | | 15.1 | 6.1 | | | | |
| **BE** | 13-15 | 74 | 49.1 | 4.6 | | 24.3 | 4.9 | | 24.8 | 4.4 | | 19.0 | 5.9 | | | | | 1.8 | 0.5 | |
| **DE** | 10-11 | 199 | 53.2 | 6.4 | 43.0-64.5 | | | | | | | | | | 17.9 | 6.0 | 9.0-28.8 | | | |
| | 12 | 114 | 52.0 | 5.4 | 42.1-60.9 | | | | | | | | | | 25.3 | 9.4 | 12.6-46.1 | | | |
| | 13-14 | 214 | 51.7 | 6.2 | 43.1-62.5 | | | | | | | | | | 27.7 | 12.2 | 11.8-50.1 | | | |
| **DK** | 10-13 | 145 | 52.0 | 4.7 | 44.0-60.0 | | | | | | | 14.0 | 5.7 | 7.0-25.0 | 18.0 | 6.3 | 9.0-28.0 | 1.9 | 0.4 | 1.3-2.6 |
| **FR** | 10-14 | 160 | 48.1 | 0.4[1] | | | | | | | | | | | 15.2 | 0.4[1] | | | | |
| **HU** | 11-14 | 124 | 50.3 | 5.6 | | | | | | | | 11.9 | 5.6 | | 20.8 | 5.9 | | | | |
| **IE** | 9-12 | 148 | 52.5 | 5.3 | 44.6-61.1 | | | | | | | | | | | | | | | |
| **IT** | 10-14 | | | | | | | | | | | | | | 21.6 | 7.6 | | | | |
| **NL** | 10-12 | 112 | 51.5 | 6.4 | 40.5-61.2 | 27.4 | 7.2 | 18.2-35.4 | 24.0 | 4.8 | 16.8-30.5 | | | | 19.0 | 6.0 | 10.0-29.0 | 2.1 | 0.6 | 1.1-3.1 |
| | 13-15 | 137 | 51.2 | 5.8 | 41.8-60.9 | 27.0 | 6.2 | 16.8-37.0 | 24.1 | 3.7 | 17.9-30.1 | | | | 22.0 | 7.0 | 11.0-34.0 | 2.0 | 0.5 | 1.0-3.0 |
| **NO** | 13 | 590 | 55.0 | 7.0 | | | | | | | | 18.0 | 8.0 | | 16.0 | 8.0 | | | | |
| **PL** | 10-14 | 202 | 53.9 | 8.0 | | | | | | | | 15.2 | 5.8 | | 24.6 | 10.0 | | | | |
| **PT** | 13 | 987 | 52.3 | 5.7 | | | | | | | | 24.2 | 6.0 | | 25.4 | 10.3 | | | | |
| **SE** | 11-12 | 517 | 52.4 | 5.6 | 43.0-61.8 | 23.4 | | | | | | 11.7 | 5.0 | 3.9-19.9 | 13.0 | 4.0 | 7.0-21.0 | 1.7 | 0.4 | 1.1-2.5 |
| **ES** | 10-14 | 66 | 41.0 | 4.2 | | | | | | | | 16.1 | 3.3 | | 18.5 | 1.6 | | | | |
| **UK** | 11-14 | 237 | 51.7 | 4.6 | 42.5-59.8 | | | | | | | | | | | | | | | |
| **Females** | | | | | | | | | | | | | | | | | | | | |
| **AT** | 10-14 | 239 | 52.1 | 8.0 | | | | | | | | 16.8 | 7.3 | | 13.7 | 4.3 | | | | |
| **BE** | 13-15 | 89 | 49.1 | 5.4 | | 24.3 | 5.0 | | 24.8 | 4.6 | | 15.8 | 5.2 | | | | | 2.0 | 0.6 | |
| **DE** | 10-11 | 198 | 53.1 | 7.2 | 43.6-66.4 | | | | | | | | | | 17.7 | 5.5 | 9.3-27.2 | | | |
| | 12 | 103 | 52.8 | 6.6 | 42.2-65.4 | | | | | | | | | | 25.0 | 10.9 | 11.0-46.5 | | | |
| | 13-14 | 230 | 52.7 | 6.1 | 44.0-63.3 | | | | | | | | | | 24.4 | 8.8 | 12.6-38.9 | | | |
| **DK** | 10-13 | 131 | 52.0 | 4.7 | | | | | | | | 14.0 | 5.0 | 7.0-23.0 | 15.0 | 5.0 | 8.0-26.0 | 1.9 | 0.5 | 1.3-2.7 |
| **FR** | 10-14 | 144 | 48.0 | 0.4[1] | | | | | | | | | | | 13.8 | 0.3[1] | | | | |
| **HU** | 11-14 | 111 | 51.7 | 5.4 | | | | | | | | 12.5 | 5.9 | | 20.1 | 6.7 | | | | |
| **IE** | 9-12 | 148 | 52.5 | 5.3 | 44.6-61.1 | | | | | | | | | | | | | | | |
| **IT** | 10-14 | 47 | | | | | | | | | | | | | 16.8 | 3.9 | | | | |
| **NL** | 10-12 | 124 | 52.1 | 6.2 | 41.2-61.7 | 28.5 | 6.2 | 18.2-38.2 | 23.6 | 4.2 | 16.9-30.1 | | | | 17.0 | 6.0 | 9.0-28.0 | 2.0 | 0.6 | 1.2-3.0 |
| | 13-15 | 117 | 50.3 | 6.5 | 40.2-62.6 | 26.4 | 7.2 | 12.3-37.8 | 23.8 | 4.6 | 17.5-32.7 | | | | 18.0 | 6.0 | 9.0-30.0 | 2.1 | 0.7 | 1.1-3.3 |
| **NO** | 13 | 515 | 55.0 | 6.0 | | | | | | | | 19.0 | 7.0 | | 14.0 | 7.0 | | | | |
| **PL** | 10-14 | 202 | 54.0 | 7.7 | | | | | | | | 15.5 | 6.0 | | 20.9 | 8.7 | | | | |

| | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **PT** | 13 | 1053 | 52.6 | 6.2 | | | | | 25.4 | 7.0 | | 25.2 | 10.6 | | | | | | |
| **SE** | 11-12 | 499 | 53.2 | 5.5 | 44.2-62.3 | 24.7 | | | 12.9 | 5.0 | 5.5-22.2 | 12.0 | 4.0 | 6-19 | | 1.8 | 0.4 | 1.2-2.5 | |
| **ES** | 10-14 | 53 | 41.6 | 3.3 | | | | | 16.0 | 2.5 | | 17.5 | 1.1 | | | | | | |
| **UK** | 11-14 | 238 | 51.2 | 5.2 | 42.2-62.8[2] | | | | | | | | | | | | | | |

[1]SE; [2] P2.5-97.5

## ANNEX 2B INTAKE OF CARBOHYDRATES AND DIETARY FIBRE AMONG CHILDREN AGED ~15-18 YEARS IN EU COUNTRIES.

| Country | Age yrs | N | Total Carbohydrates E% | | | Mono- and Disaccharides (E%) | | | Polysaccharides (E%) | | | Sucrose (E%) | | | Dietary Fibre (g) | | | Dietary Fibre (g/MJ) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 |
| **Males** | | | | | | | | | | | | | | | | | | | | |
| **AT** | 14->19 | 1527 | 46.1 | 9.9 | | | | | | | | 16.1 | 8.9 | | 15.6 | 7.1 | | | | |
| **BE** | 15-18 | 405 | 49.5 | 4.4 | | | | | | | | | | | | | | | | |
| **DE** | 15-17 | 294 | 49.6 | 6.5 | | | | | | | | | | | 26.1 | 10.7 | | | | |
| **DK** | 14-17 | 86 | 50.0 | 6.1 | 41-59 | | | | | | | 13.0 | 7.2 | 2-27 | 19.0 | 6.0 | 9-28 | 1.8 | 0.5 | 1.1-2.6 |
| **FR** | 15-18 | 181 | 48.7 | 0.6[1] | | | | | | | | | | | 16.9 | 0.8[1] | | | | |
| **IT** | 15-18 | 52 | | | | | | | | | | | | | 23.9 | 9.1 | | | | |
| **NL** | 16-18 | 142 | 49.5 | 6.6 | 38.7-59.9 | 24.9 | 7.4 | 14.5-35.7 | 24.5 | 4.8 | 16.5-32.6 | | | | 24.0 | 11.0 | 8-42 | 2.1 | 0.7 | 1.0-3.3 |
| **NO** | 16-19 | 92 | 53.7 | | | | | | | | | 15.1 | | | 26.0 | | | | | |
| **PL** | 15-18 | 174 | 50.7 | 7.4 | | | | | | | | 12.5 | 5.0 | | 32.6 | 12.5 | | | | |
| **SI** | 15-18 | 1010 | 57.0 | 9.0 | | | | | | | | 11.0 | 4.0 | | 33.0 | 21.0 | | | | |
| **ES** | 15-18 | 61 | 39.7 | 4.7 | | | | | | | | 14.8 | 3.4 | | 18.9 | 1.8 | | | | |
| **UK** | 15-18 | 179 | 50.5 | 5.4 | 39.9-60.5 | | | | | | | | | | | | | | | |
| **Females** | | | | | | | | | | | | | | | | | | | | |
| **AT** | 14->19 | 1422 | 47.3 | 10.3 | | | | | | | | 15.7 | 8.7 | | 13.8 | 6.0 | | | | |
| **BE** | 15-18 | 401 | 50.6 | 5.4 | | | | | | | | | | | | | | | | |
| **DE** | 15-17 | 317 | 52.7 | 6.4 | | | | | | | | | | | 23.1 | 8.3 | | | | |
| **DK** | 14-17 | 117 | 52.0 | 5.6 | 45-63 | | | | | | | 14.0 | 6.1 | 5-23 | 15.0 | 5.2 | 8-24 | 1.9 | 0.5 | 1.2-2.6 |
| **FR** | 15-18 | 222 | 48.8 | 0.7[1] | | | | | | | | | | | 12.7 | 0.3[1] | | | | |
| **IT** | 15-18 | 47 | | | | | | | | | | | | | 17.6 | 4.7 | | | | |
| **NL** | 16-18 | 129 | 50.3 | | 37.6-61.1 | 26.2 | | 12.8-37.5 | 24.0 | | 16.2-32.9 | | | | 19.0 | | 8-20 | 2.1 | | 1.1-3.6 |
| **NO** | 16-19 | 62 | 54.6 | | | | | | | | | 11.7 | | | 21.0 | 8.0 | | | | |
| **PL** | 15-18 | 175 | 54.2 | 8.5 | | | | | | | | 14.1 | 5.7 | | 23.0 | 8.9 | | | | |
| **SI** | 15-18 | 1214 | 57.0 | 8.0 | | | | | | | | 13.0 | 4.0 | | 27.0 | 18.0 | | | | |
| **ES** | 15-18 | 57 | 38.6 | 3.7 | | | | | | | | 15.4 | 2.9 | | 16.2 | 2.0 | | | | |
| **UK** | 15-18 | 210 | 50.6 | 5.6 | 39.9-64.0[2] | | | | | | | | | | | | | | | |

[1]SE; [2] P2.5-97.5

## ANNEX 3A POPULATION, METHODS AND PERIOD OF DIETARY ASSESSMENT IN ADULTS IN EUROPEAN COUNTRIES.

| Country | Population | Dietary method | Year of survey | Reference |
|---|---|---|---|---|
| **AT** | Males and females aged 19-64 years | 24-hour recall | 2007 | Elmadfa et al., 2009 |
| | Males and females aged 65 and over | 3-day record | 2007 | Elmadfa et al., 2009 |
| **BE** | Males and females aged 19-59 years | 2x 24-hour recall | 2004 | De Vriese et al, 2006 |
| | Males and females aged 60-75 years | 2x 24-hour recall | 2004 | De Vriese et al, 2006 |
| | Males and females aged 75+ years | 2x 24-hour recall | 2004 | De Vriese et al, 2006 |
| **CZ** | Males and females aged 19-64 years | n.a. | n.a.[1] | Cifkova and Skodova, 2004 |
| **DE** | Males and females aged 35-64 years | 24-hour recall | 1996-1998 | Linseisen et al., 2003 |
| | Males and females aged 19-64 years | 24-hour recall + Dietary History | 2005-2006 | Anonymous, 2008 |
| | Males and females aged 19-24 years | 24-hour recall + Dietary History | 2005-2006 | Anonymous, 2008 |
| | Males and females aged 25-34 years | 24-hour recall + Dietary History | 2005-2006 | Anonymous, 2008 |
| | Males and females aged 35-50 years | 24-hour recall + Dietary History | 2005-2006 | Anonymous, 2008 |
| | Males and females aged 51-64 years | 24-hour recall + Dietary History | 2005-2006 | Anonymous, 2008 |
| | Males and females aged 65-80 years | 24-hour recall + Dietary History | 2005-2006 | Anonymous, 2008 |
| | Males and Females aged 65 and over | 24-hour recall + Dietary History | 2005-2006 | Anonymous, 2008 |
| **DK** | Males and females aged 18-74 years | 7-day record | 2000-2002 | Lyhne et al., 2005 |
| | Males and females aged 18-24 years | 7-day record | 2000-2002 | Lyhne et al., 2005 |
| | Males and females aged 25-34 years | 7-day record | 2000-2002 | Lyhne et al., 2005 |
| | Males and females aged 35-44 years | 7-day record | 2000-2002 | Lyhne et al., 2005 |
| | Males and females aged 45-54 years | 7-day record | 2000-2002 | Lyhne et al., 2005 |
| | Males and females aged 55-64 years | 7-day record | 2000-2002 | Lyhne et al., 2005 |
| | Males and females aged 65-74 years | 7-day record | 2000-2002 | Lyhne et al., 2005 |
| **EE** | Males and females aged 19-65 years | 24-hour recall | 1997 | Pomerleau et al., 2001 |
| | Males and females aged 19-34 years | 24-hour recall | 1997 | Pomerleau et al., 2001 |
| | Males and females aged 35-49 years | 24-hour recall | 1997 | Pomerleau et al., 2001 |
| | Males and females aged 50 and over | 24-hour recall | 1997 | Pomerleau et al., 2001 |
| **FI** | Males and females aged 25-64 years | 3-day record | 2002 | Paturi et al., 2008 |
| | Males and females aged 65-74 years | 4-day record | 2002 | Paturi et al., 2008 |
| **FR** | Males and females aged 19-64 years | 3x 24-hour recall | 2006-2007 | Castetbon et al. 2009. (In: Elmadfa, 2009) |
| | Males and females aged 65-75 years | 3x 24-hour recall | 2006-2007 | Castetbon et al. 2009. (In: Elmadfa, 2009) |

| | | | | |
|---|---|---|---|---|
| **GR** | Males and females aged 19-64 years | FFQ + 24-hour recall in sub group | 1994-1999 | Greek cohort EPIC study. (In: Elmadfa, 2009) |
| | Males and females aged 65 and over | FFQ | 1994-1999 | Greek cohort EPIC study. (In: Elmadfa, 2009) |
| **HU** | Males and females aged 11-14 years | 3-day record | 2003-2004 | Rodler et al. 2005; Zajkás et al., 2007; Bíró et al., 2007 (In: Elmadfa, 2009) |
| | Males and females aged 18-59 | 3-day record | 2003-2004 | Rodler et al. 2005; Zajkás et al., 2007; Bíró et al., 2007 (In: Elmadfa, 2009) |
| | Males and females aged 60 and over | 3-day record | 2003-2004 | Rodler et al. 2005; Zajkás et al., 2007; Bíró et al., 2007 (In: Elmadfa, 2009) |
| **IE** | Males and females 18-64 years | 7-day record | 1997-1999 | Irish Universities Nutrition Alliance, North/South Ireland Food Consumption Survey. www.iuna.net |
| | Males and females 18-35 years | 7-day record | 1997-1999 | Irish Universities Nutrition Alliance, North/South Ireland Food Consumption Survey. www.iuna.net |
| | Males and females 36-50 years | 7-day record | 1997-1999 | Irish Universities Nutrition Alliance ,North/South Ireland Food Consumption Survey. www.iuna.net |
| | Males and females 51-64 years | 7-day record | 1997-1999 | Irish Universities Nutrition, Alliance North/South Ireland Food Consumption Survey. www.iuna.net |
| **IT** | Males and females 19-64 years | 7-day record | n.a. | D"Amicis, 2000 |
| | Males and females aged 65 and over | 7-day record | n.a. | D"Amicis, 2000 |
| **LT** | Males and females 19-64 years | 24-hour recall | 2007 | Unpublished data (In: Elmadfa, 2009) |
| **LV** | Males and females 19-64 years | 24-hour recall | 1997 | Pomerleau et al., 2001 |
| | Males and females aged 19-34 years | 24-hour recall | 1997 | Pomerleau et al., 2001 |
| | Males and females aged 35-49 years | 24-hour recall | 1997 | Pomerleau et al., 2001 |
| | Males and females aged 50 and over | 24-hour recall | 1997 | Pomerleau et al., 2001 |
| **NL** | Males and Females aged 19-64 years | 2-day record | 1997-1998 | Hulshof et al., 1998 |
| | Males and Females aged 65 and over | 2-day record | 1997-1998 | Hulshof et al., 1998 |
| | Males and females aged 19-30 years | 2x 24-hour recall | 2003 | Hulshof and Ocké, 2005 |
| **NO** | Males and females aged 19-64 years | FFQ | 1997 | Johansson and Sovoll, 1999 |
| | Males and females aged 65 and over | FFQ | 1997 | Johansson and Sovoll, 1999 |
| **PL** | Males and females aged 19-64 years | 24-hour recall | 2000 | Szponar et al., 2000 unpublished data (In: Elmadfa, 2009) |
| | Males and females aged 65 and over | 24-hour recall | 2000 | Szponar et al., 2000 unpublished data (In: Elmadfa, 2009) |
| **PT** | Males and females aged 18+ years | FFQ | n.a. | EPIPorto study (In: Elmadfa, 2009) |
| | Males and females aged 65 and over | FFQ | n.a. | EPIPorto study (In: Elmadfa, 2009) |

| | | | | |
|---|---|---|---|---|
| **RO** | Males and females aged 19-64 years | personal interview | 2006 | National Synthesis 2006 (In: Elmadfa, 2009) |
| | Males and females aged 65 and over | personal interview | 2006 | National Synthesis 2006 (In: Elmadfa, 2009) |
| **ES** | Males and females aged 18-64 years | 24-hour recall | 2002-2003 | Serra Majem et al., 2007 |
| | Males and females aged 65-75 years | 24-hour recall | 2002-2003 | Serra Majem et al., 2007 |
| **SE** | Males and females aged 18-74 years | 7-day record | 1997-1998 | Becker and Pearson, 2002 |
| | Males and females aged 17-24 years | 7-day record | 1997-1998 | Becker and Pearson, 2002 |
| | Males and females aged 25-34 years | 7-day record | 1997-1998 | Becker and Pearson, 2002 |
| | Males and females aged 35-44 years | 7-day record | 1997-1998 | Becker and Pearson, 2002 |
| | Males and females aged 45-54 years | 7-day record | 1997-1998 | Becker and Pearson, 2002 |
| | Males and females aged 55-64 years | 7-day record | 1997-1998 | Becker and Pearson, 2002 |
| | Males and females aged 65 and over | 7-day record | 1997-1998 | Becker and Pearson, 2002 |
| **UK** | Males and females aged 19-64 years | 7-day record | 2000-2001 | Henderson et al., 2003 |
| | Males and females aged 19-24 years | 7-day record | 2000-2001 | Henderson et al., 2003 |
| | Males and females aged 25-34 years | 7-day record | 2000-2001 | Henderson et al., 2003 |
| | Males and females aged 35-49 years | 7-day record | 2000-2001 | Henderson et al., 2003 |
| | Males and females aged 50-64 years | 7-day record | 2000-2001 | Henderson et al., 2003 |
| | Males and females aged 65+ years | 4-day record | 1994-1995 | Finch et al., 1998 |

[1]n.a. = not available

## ANNEX 3B INTAKE OF CARBOHYDRATES AND DIETARY FIBRE AMONG ADULTS AGED ~19-65 YEARS IN EU COUNTRIES.

| Country | Age yrs | N | Total Carbohydrates (E%) | | | Mono- and Disaccharides (E%) | | | Polysaccharides (E%) | | | Sucrose (E%) | | | Dietary Fibre (g) | | | Dietary Fibre (g/MJ) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 |
| **Males** | | | | | | | | | | | | | | | | | | | | |
| **AT** | 19-64 | 778 | 42.5 | 10.5 | | | | | | | | 8.7 | 6.4 | | 19.5 | 9.9 | | | | |
| **BE** | 19-59 | 413 | 45.2 | 5.3 | | 24.1 | 4.8 | | 19.4 | 5.7 | | | | | | | | | | |
| **CZ** | 19-64 | 1094 | 53.9 | 12.0 | | | | | | | | | | | | | | | | |
| **DE** | 19-64 | 4912 | 45.3 | 8.4 | | | | | | | | | | | | | | | | |
| **DK** | 18-74 | 1467 | 43 | 6.8 | 35.0-53.0[1] | | | | | | | 8.0 | | 3-15[1] | 21.0 | | 12.0-32.0[1] | | | |
| **EE** | 19-64 | 900 | 42.7 | 14.0 | | | | | | | | | | | | | | | | |
| **FI** | 25-64 | 730 | 47.1 | 8.8 | | | | | | | | 9.7 | 5.9 | | 24.0 | 11.0 | | 2.7 | 1.1 | |
| **FR** | 19-64 | 852 | 43.4 | 0.3[2] | | | | | | | | | | | 18.7 | 0.4[2] | | | | |
| **GR** | 19-64 | 8365 | 37.9 | 5.9 | | | | | | | | | | | | | | | | |
| **HU** | >18 | 473 | 45.0 | 6.6 | | | | | | | | 7.6 | 5.2 | | 24.2 | 6.6 | | | | |
| **IE** | 18-64 | 662 | 43.5 | 6.4 | 32.6-54.3 | | | | | | | | | | 23.2 | 8.5 | 12.1-38.9 | | | |
| **IT** | 19-64 | 660 | | | | | | | | | | | | | 21.8 | 6.5 | | | | |
| **LT** | 19-65 | 849 | 38.9 | 9.3 | | | | | | | | 10.8 | 5.6 | | 20.9 | 12.4 | | | | |
| **LV** | 19-65 | 1065 | 42.2 | 11.8 | | | | | | | | | | | | | | | | |
| **NL** | 19-64 | 1836 | 44.2 | 7.5 | | | | | | | | | | | | | | | | |
| **NO** | 19-64 | 1050 | 51.0 | 6.0 | | | | | | | | 9.0 | 6.0 | | 25.0 | 10.0 | | | | |
| **PL** | 19-64 | 1106 | 48.0 | 8.4 | | | | | | | | 11.0 | 5.2 | | 29.7 | 11.4 | | | | |
| **PT** | 19-64 | 917 | 47.0 | 6.8 | | | | | | | | | | | 23.5 | 9.0 | | | | |
| **RO** | 19-64 | 177 | 43.3 | 9.4 | | | | | | | | | | | | | | | | |
| **SE** | 18-74 | 589 | 46 | 6.0 | 37.0-56.0 | | | | | | | 9.0 | 4.0 | 3-16 | 18.0 | 7.0 | 9.0-29.0 | 1.8 | 0.5 | 1.1-2.7 |
| **ES** | 18-64 | 718 | 40.5 | | | 16.6 | | | 23.2 | | | | | | 19.2 | | | | | |
| **UK** | 19-64 | 833 | 47.7 | 6.0 | 35.9-59.8[4] | | | | | | | | | | | | | | | |
| **Females** | | | | | | | | | | | | | | | | | | | | |
| **AT** | 19-64 | 1345 | 46.0 | 10.6 | | | | | | | | 10.9 | 6.6 | | 20.1 | 9.3 | | | | |
| **BE** | 19-59 | 460 | 46.9 | 6.2 | | 21.4 | 6.3 | | 24.3 | 4.1 | | | | | | | | | | |
| **CZ** | 19-64 | 1094 | 53.9 | 12.0 | | | | | | | | | | | | 9.0 | | | | |
| **DE** | 19-64 | 6016 | 48.7 | 7.4 | | | | | | | | | | | | | | | | |
| **DK** | 18-74 | 1684 | 47 | 6.6 | 39.0-55.0[1] | | | | | | | 9.0 | | 4-17[1] | 19.0 | | 11.0-28.0[1] | | | |
| **EE** | 19-65 | 1115 | 47.3 | 12.6 | | | | | | | | | | | | | | | | |
| **FI** | 25-64 | 846 | 50.2 | 8.3 | | | | | | | | 10.5 | 5.1 | | 21.0 | 9.0 | | 3.2 | 1.3 | |
| **FR** | 19-64 | 1499 | 44.4 | 0.2[2] | | | | | | | | | | | 15.7 | 0.2[2] | | | | |
| **GR** | 19-64 | 12034 | 39.5 | 5.4 | | | | | | | | | | | | | | | | |
| **HU** | >18 | 706 | 48.0 | 5.8 | | | | | | | | 8.6 | 4.8 | | 21.7 | 5.6 | | | | |
| **IE** | 18-64 | 717 | 46.6 | 5.6 | 37.4-56.6 | | | | | | | | | | 17.4 | 5.9 | 9.3-27.5 | | | |
| **IT** | 19-64 | 801 | | | | | | | | | | | | | 18.9 | 6.1 | | | | |

| | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **LT** | 19-64 | 1087 | 42.9 | 10.3 | | | | | | | | | | | | | | | | | | |
| **LV** | 19-64 | 1235 | 44.6 | 11.9 | | | | | | | | | | | | | | | | | | |
| **NL** | 19-64 | 2112 | 44.7 | 7.9 | | | | | | | | | | | | | | | | | | |
| **NO** | 19-64 | 1146 | 51.0 | 6.0 | | | | | | | | | 9.0 | 6.0 | | | 21.0 | 8.0 | | | | |
| **PL** | 19-64 | 1334 | 51.8 | 9.1 | | | | | | | | | 13.7 | 6.6 | | | 19.7 | 7.9 | | | | |
| **PT** | 19-64 | 1472 | 50.1 | 5.9 | | | | | | | | | | | | | 23.7 | 9.4 | | | | |
| **RO** | 19-64 | 341 | 43.6 | 9.7 | | | | | | | | | | | | | | | | | | |
| **SE** | 18-74 | 626 | 47 | 5.0 | | 20.0 | | | | | | | 9.0 | 4.0 | 4-16 | | 16.0 | 5.0 | 9-27 | | 2.1 | 0.5 | 1.3-3.1 |
| **ES** | 18-64 | 895 | 40.7 | | | 18.5 | | 21.0 | | | | | | | | | 16.9 | | | | | |
| **UK** | 19-64 | 891 | 48.5 | 6.7 | 37.4-61.5[3] | | | | | | | | | | | | | | | | | |

[1]P10-P90; [2] SE; [3] P2.5-P97.5

## ANNEX 3B INTAKE OF CARBOHYDRATES AND DIETARY FIBRE AMONG ADULTS AGED ~19-34 YEARS IN EU COUNTRIES.

| Country | Age yrs | N | Total Carbohydrates (E%) | | | Mono- and Disaccharides (E%) | | | Polysaccharides (E%) | | | Sucrose (E%) | | | Dietary Fibre (g) | | | Dietary Fibre (g/MJ) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 |
| **Males** | | | | | | | | | | | | | | | | | | | | |
| **DE** | 19-24 | 510 | 46.7 | 0.35[1] | 34.8-60.4 | | | | | | | | | | 24.6 | 0.55[1] | 10.1-51.4 | | | |
| | 25-34 | 690 | 46.1 | 0.29[1] | 34.4-59.4 | | | | | | | | | | 25.8 | 0.44[1] | 10.5-46.1 | | | |
| **DK** | 18-25 | 146 | 47.0 | 5.8 | 39.0-58.0 | | | | | | | 12 | 6.5 | 3-25 | 19.0 | 7.3 | 10.0-32.0 | 1.8 | 0.6 | 1.1-2.9 |
| | 25-34 | 272 | 46.0 | 5.4 | 37.0-55.0 | | | | | | | 11 | 5.7 | 3-20 | 20.0 | 6.9 | 11.0-32.0 | 1.9 | 0.5 | 1.2-2.8 |
| **EE** | 19-34 | 396 | 41.8 | 131 | | | | | | | | | | | | | | | | |
| **IE** | 18-35 | 253 | 42.7 | 6.2 | 31.9-53.3 | | | | | | | | | | 22.6 | 8.5 | 11.7-38.5 | | | |
| **LV** | 19-34 | 337 | 41.3 | 11.2 | | | | | | | | | | | | | | | | |
| **NL** | 19-30 | 352 | 47.5 | 4.3 | 40.4-54.6 | 23.8 | 5.2 | 15.6-32.8 | | | | | | | 22.7 | 6.1 | 13.6-33.4 | 2.0 | 0.5 | 1.2-2.9 |
| **SE** | 17-24 | 67 | 49.0 | 5.0 | 40.0-57.0 | | | | | | | 11 | 5.0 | 4-21 | 16.0 | 7.0 | 6.0-29.0 | 1.6 | 0.4 | 1.0-2.5 |
| | 25-34 | 128 | 47.0 | 6.0 | 38.0-57.0 | | | | | | | 10 | 4.0 | 4-17 | 18.0 | 6.0 | 9.0-28.0 | 1.7 | 0.5 | 1.1-2.5 |
| **UK** | 19-24 | 108 | 49.0 | 6.3 | 38.0-63.2 | | | | | | | | | | | | | | | |
| | 25-34 | 219 | 47.7 | 5.8 | 35.2-58.3 | | | | | | | | | | | | | | | |
| | **Females** | | | | | | | | | | | | | | | | | | | |
| **DE** | 19-24 | 510 | 51.3 | 0.34[1] | | | | | | | | | | | 21.7 | 0.39[3] | 8.7-37.1 | | | |
| | 25-34 | 972 | 50.0 | 0.23[1] | | | | | | | | | | | 24.0 | 0.31 | 11.9-41.1 | | | |
| **DK** | 18-25 | 213 | 50.0 | 5.5 | 42.0-61.0 | | | | | | | 13 | 6.9 | 4-26 | 16.0 | 4.5 | 9.0-22.0 | 2.0 | 0.6 | 1.2-3.2 |
| | 25-34 | 315 | 49.0 | 5.4 | 40.0-57.0 | | | | | | | 12 | 5.8 | 4-23 | 17.0 | 4.5 | 10.0-25.0 | 2.1 | 0.7 | 1.2-3.3 |
| **EE** | 19-34 | 459 | 46.4 | 13.0 | | | | | | | | | | | | | | | | |
| **IE** | 18-35 | 269 | 46.6 | 5.1 | 39.0-55.7 | | | | | | | | | | 16.1 | 5.1 | 8.8-25.7 | | | |
| **LV** | 19-34 | 342 | 44.3 | 12.1 | | | | | | | | | | | | | | | | |
| **NL** | 19-30 | 398 | 49.3 | 5.5 | 40.5-58.4 | 25.6 | 6.1 | 16.3-36.3 | | | | | | | 17.0 | 4.5 | 10.2-25.0 | 2.2 | 0.6 | 1.3-3.3 |
| **SE** | 17-24 | 70 | 50.0 | 6.0 | 39.0-61.0 | | | | | | | 11 | 4.0 | 5-18 | 15.0 | 5.0 | 7.0-24.0 | 1.9 | 0.5 | 1.2-2.9 |
| | 25-34 | 132 | 48.0 | 5.0 | 40.0-56.0 | | | | | | | 10 | 4.0 | 5-16 | 15.0 | 4.0 | 9.0-23.0 | 1.9 | 0.5 | 1.2-2.8 |
| **UK** | 19-24 | 104 | 49.1 | 8.3 | 36.5-62.7[2] | | | | | | | | | | | | | | | |
| | 25-34 | 210 | 48.7 | 5.8 | 36.6-61.9[2] | | | | | | | | | | | | | | | |

[1]SE; [2] P2.5-P97.5

## ANNEX 3B INTAKE OF CARBOHYDRATES AND DIETARY FIBRE AMONG ADULTS AGED ~35-64 YEARS IN EU COUNTRIES.

| Country | Age yrs | N | Total Carbohydrates (E%) | | | Mono- and Disaccharides (E%) | | | Polysaccharides (E%) | | | Sucrose (E%) | | | Dietary Fibre (g) | | | Dietary Fibre (g/MJ) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 | *mean* | *SD* | P5 - P95 |
| **Males** | | | | | | | | | | | | | | | | | | | | |
| **DE** | 35-64[1] | 1013 | 40.2 | 9.9 | | 17.6 | | | 22.4 | | | 8.8 | 6.0 | | 21.8 | 11.5 | | | | |
| | 35-64[2] | 1032 | 38.5 | 9.4 | | 17.4 | | | 20.8 | | | 8.9 | 5.7 | | 21.9 | 9.2 | | | | |
| | 35-50 | 2079 | 45.1 | 0.17[3] | 32.0-58.0 | | | | | | | | | | 27.3 | 0.26[3] | 11.9-48.2 | | | |
| | 51-64 | 1633 | 44.4 | 0.19[3] | 32.4-57.2 | | | | | | | | | | 27.4 | 0.28[3] | 12.4-48.2 | | | |
| **DK** | 35-44 | 330 | 43.0 | 5.6 | 34.0-53.0 | | | | | | | 8.0 | 4.8 | 2-18 | 22.0 | 7.6 | 11-34 | 2.0 | 0.5 | 1.3-2.9 |
| | 45-54 | 312 | 42.0 | 6.6 | 31.0-53.0 | | | | | | | 7.0 | 4.4 | 1-15 | 22.0 | 9.0 | 10-39 | 2.2 | 0.6 | 1.3-3.4 |
| | 55-64 | 242 | 42.0 | 6.9 | 31.0-53.0 | | | | | | | 6.0 | 4.1 | 1-14 | 21.0 | 8.3 | 10-37 | 2.2 | 0.7 | 1.2-3.4 |
| **EE** | 35-49 | 319 | 43.6 | 15.5 | | | | | | | | | | | | | | | | |
| | 50+ | 185 | 43.1 | 13.0 | | | | | | | | | | | | | | | | |
| **IE** | 36-50 | 236 | 43.3 | 6.3 | 32.2-53.9 | | | | | | | | | | 23.6 | 8.1 | 12.8-38.8 | | | |
| | 51-64 | 173 | 45.2 | 6.8 | 32.9-55.9 | | | | | | | | | | 17.3 | 7.1 | 9.1-31.5 | | | |
| **LV** | 35-49 | 372 | 42.6 | 11.8 | | | | | | | | | | | | | | | | |
| | 50+ | 356 | 43.2 | 12.4 | | | | | | | | | | | | | | | | |
| **SE** | 35-44 | 143 | 45.0 | 5.0 | 38.0-54.0 | | | | | | | 8.0 | 4.0 | 3-14 | 18.0 | 7.0 | 9.0-30.0 | 1.8 | 0.4 | 1.2-2.5 |
| | 45-54 | 18 | 46.0 | 6.0 | 36.0-56.0 | | | | | | | 8.0 | 4.0 | 3-16 | 19.0 | 7.0 | 9.0-34.0 | 1.9 | 0.6 | 1.2-2.8 |
| | 55-64 | 68 | 47.0 | 5.0 | 39.0-55.0 | | | | | | | 7.0 | 4.0 | 3-14 | 18.0 | 5.0 | 12.0-29.0 | 2.0 | 0.4 | 1.5-2.8 |
| **UK** | 35-49 | 253 | 47.5 | 5.9 | 36.0-59.9[4] | | | | | | | | | | | | | | | |
| | 50-64 | 253 | 47.4 | 6.2 | 35.6-59.5[4] | | | | | | | | | | | | | | | |
| **Females** | | | | | | | | | | | | | | | | | | | | |
| **DE** | 35-64[1] | 1078 | 43.0 | 10.2 | | 21.5 | | | 21.1 | | | 10.9 | 6.6 | | 19.6 | 9.0 | | | | |
| | 35-64[2] | 898 | 43.9 | 10.6 | | 23.3 | | | 20.1 | | | 12.1 | 7.3 | | 19.4 | 8.2 | | | | |
| | 35-50 | 2694 | 48.0 | 0.14 | | | | | | | | | | | 24.7 | 0.19 | 11.5-42.9 | | | |
| | 51-64 | 1840 | 47.7 | 0.18 | | | | | | | | | | | 26.1 | 0.24 | 12.7-44.2 | | | |
| **EE** | 35-49 | 376 | 47.4 | 12.4 | | | | | | | | | | | | | | | | |
| | 50+ | 280 | 48.5 | 12.3 | | | | | | | | | | | | | | | | |
| **DK** | 35-44 | 359 | 47.0 | 5.9 | 38.0-57.0 | | | | | | | 9.0 | 5.4 | 3-18 | 15.0 | 5.0 | 8.0-26.0 | 2.3 | 0.7 | 1.4-3.5 |
| | 45-54 | 370 | 46.0 | 6.1 | 36.0-56.0 | | | | | | | 7.0 | 4.1 | 2-15 | 15.0 | 5.2 | 8.0-24.0 | 2.5 | 0.6 | 1.6-3.6 |
| | 55-64 | 263 | 46.0 | 6.5 | 35.0-56.0 | | | | | | | 8.0 | 4.1 | 2-14 | 16.0 | 5.6 | 9.0-27.0 | 2.6 | 0.7 | 1.6-3.9 |
| **IE** | 36-50 | 286 | 44.7 | 6.1 | 33.9-54.1 | | | | | | | | | | 18.2 | 5.3 | 10.7-28.5 | | | |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 51-64 | 162 | 47.5 | 6.2 | 37.3-57.6 | | | | | | 18.2 | 6.2 | 8.8-28.2 | | | | |
| **LV** | 35-49 | 396 | 43.4 | 11.7 | | | | | | | | | | | | | |
| | 50+ | 496 | 45.7 | 12.0 | | | | | | | | | | | | | |
| **SE** | 35-44 | 132 | 46.0 | 6.0 | 37.0-54.0 | | | 9.0 | 3.0 | 4-14 | 16.0 | 4.0 | 9.0-22.0 | 2.0 | 0.5 | 1.4-2.9 | |
| | 45-54 | 153 | 47.0 | 5.0 | 39.0-55.0 | | | 8.0 | 3.0 | 4-14 | 17.0 | 5.0 | 10.0-27.0 | 2.2 | 0.5 | 1.4-3.3 | |
| | 55-64 | 81 | 48.0 | 5.0 | 40.0-57.0 | | | 8.0 | 4.0 | 4-13 | 18.0 | 5.0 | 10.0-28.0 | 2.4 | 0.5 | 1.7-3.5 | |
| **UK** | 35-49 | 318 | 48.6 | 6.8 | 35.3-62.4[4] | | | | | | | | | | | | |
| | 50-64 | 259 | 48.1 | 6.7 | 32.2-59.4[4] | | | | | | | | | | | | |

[1]Cohort Heidelberg; [2]Cohort Potsdam; [3]SE ; [4] P2.5-P97.5;

## ANNEX 4 EFFECTS OF SUGAR INTAKE ON GLUCOSE AND INSULIN RESPONSE IN CONTROLLED INTERVENTION STUDIES IN ADULTS.

| Study | Study design | Subjects | Total fat, E% | CHO, E% | Sugars, E% | Results |
|---|---|---|---|---|---|---|
| Reiser et al. 1979 | 6-week cross-over, iso-energetic | 10 men, 9 women, 35-55 y | 42 | 43 | 30 E% sucrose or wheat starch | Fasting serum glucose and insulin higher on sucrose diet |
| Reiser et al 1981 | 6-week cross-over, iso-energetic | 24 adults with impaired insulin response | 42 | 44 | 5, 18 or 33 E% sucrose or starch | Fasting and postprandial insulin higher on 18 or 33 E% sucrose diets |
| Swanson et al. 1992 | 4-week cross-over, iso-energetic | 14 healthy adults | 32-34 | 51-52 | 20 E% as fructose or starch (< 3 E% fructose) | No sign. differences between the diets in mean values of hemoglobin A1c, serum glycosylated albumin, fasting plasma glucose, peak postprandial plasma glucose, or integrated plasma glucose response. |
| Bantle et al., 2000 | 6-week cross-over, iso-energetic | 12 men, 12 women (BMI <32) | 30 | 55 | 17 E% fructose or glucose. Total sugars (glucose, fructose, sucrose and lactose) about 21 E% in both diets | Lower postprandial plasma glucose serum insulin, and daylong insulin concentrations on fructose diet |
| Black et al. 2006 | 6 wk cross-over, iso-energetic | 13 W/M 33 y, BMI: 26.6 | 33 | 55 | 10 or 25 E% sucrose | No difference in glucose serum insulin or peripheral insulin resistance |

## ANNEX 5 EFFECTS OF SUGAR INTAKE ON SERUM LIPIDS IN CONTROLLED INTERVENTION STUDIES IN ADULTS

| Study | Study design | Subjects | Total fat (E%) | CHO (E%) | Sugars (E%) | Results |
|---|---|---|---|---|---|---|
| **Short-term** | | | | | | |
| Hallfrisch et al., 1983 | 5-week cross-over | 12 men hyper-insulinemic 12 with normal insulin response | | | 0, 7.5, and 15 E% fructose replacing starch | Total and LDL-chol +5-7% (sign) in both groups on 7.5 E% and 15 E% fructose diets. Fasting TG +30% and +61%(sign) on 7.5 and 15 E% fructose diets, respectively |
| Reiser et al. 1989 | 5-week cross-over | 10 hyper-insulemic and 11 normal men | 36 | 51 | 20 E% as either added fructose or high-amylose cornstarch | Fasting TG (+46%), total chol (+11%) in the hyperinsulinemics, and +20% and +8%, respectively, in normal men on the fructose diet. LDL-chol (+12%) in the normal men on the fructose diet |
| Swanson et al., 1992 | 4-week cross-over | 14 healthy subjects | 30 | 55 | 20 E% added fructose or starch | No difference in fasting serum TG. Total and LDL chol +9% and +11% (sign), respectively, on fructose diet |
| Bantle et al. 2000 | 6-week cross-over | 12 men and 12 women BMI < 32 | | | 17 E% as fructose or glucose, Total sugars 21E% | Increased fasting and daylong TG in men, but not in women, on fructose diet. Total and LDL-chol higher on day 28, but not at the end of the study period on the fructose diet. |
| Marckmann et al. 2000 | 2 wk cross-over *ad libitum*. | 20 W, 21-52 y BMI: | 28-29 | 59 | 2.5 or 23 E% sucrose | Fasting and nonfasting TG, total and LDL-chol lower on the low-sucrose diet. |
| Black et al. 2006 | 6 wk cross-over, iso-energetic | 13 W/M 33 y, BMI: 26.6 | | | 10 or 25 E% sucrose | Total and LDL chol +15% and +24%, respectively on high sucrose diet. HDL-chol and TG unchanged. |
| Erkkila et al. 2007 | 8 wk | 34 | | | Increased sucrose intake from 7-9 to 15 E% (40 g/day) | No significant effects on lipid concentrations |
| **Long-term** | | | | | | |
| Saris et al. 2000 | 6-month parallel, *ad libitum* | 316 obese | | | Control: 46 E% CHO (21 E% sugars) Intervention diets: 52–56 E% CHO, starch (16 E% sugars) or high-sugar (sucrose, fructose and lactose, 30 E%). | No significant changes were seen in serum lipids among the groups |
| Poppitt et al., 2002 | 6 months | 46 obese subjects with the metabolic syndrome | | | 3 diets, "complex carbohydrate", 21 or 29 E% sugars (as sucrose, fructose and lactose) | Fasting TG higher in 29 E% sugar group than in the "complex-carbohydrate" and 21 E% sugar diet. Weight loss correlated with a decrease in TG concentrations. |

## GLOSSARY / ABBREVIATIONS

| | |
|---|---|
| Added sugars | Term used to describe sucrose, fructose, glucose, starch hydrolysates (glucose syrup, high-fructose syrup) and other isolated sugar preparations used as such or added during food preparation and manufacturing |
| AFSSA | Agence Française de Sécurité Sanitaire des Aliments |
| AICR | American Institute of Cancer Research |
| AI | Adequate Intake |
| AMDR | Acceptable Macronutrient Distribution Ranges |
| AOAC | Association of Official Analytical Chemists |
| BMI | Body Mass Index |
| CHD | Coronary Heart Disease |
| CV | Coefficient of Variation |
| DMFT | Decayed, Missing and Filled Teeth in the permanent teeth |
| dmft | Decayed, missing and filled teeth in the primary teeth |
| CVD | Cardiovascular Disease |
| DoH | Department of Health |
| DP | Degree of Polymerisation |
| DRVs | Dietary Reference Values |
| EC | European Commission |
| EFSA | European Food Safety Authority |
| EPIC | European Prospective Investigation into Cancer and Nutrition |
| ESPGHAN | European Society of Paediatric Gastroenterology, Hepatology and Nutrition |
| EU | European Union |
| FAO | Food and Agriculture Organisation of the United Nations |
| FFQ | Food Frequency Questionnaire |
| FNB | U.S. Food and Nutrition Board |
| FOS | Fructo-oligosaccharides |
| GI | Glycaemic Index |

| | |
|---|---|
| GL | Glycaemic Load |
| GLUT | Glucose Transporter |
| GOS | Galacto-oligosaccharides |
| HDL-cholesterol | High Density Lipoprotein-cholesterol |
| HOMA-IR | Homeostasis Model Assessment of Insulin Resistance |
| HOMA-ß | Homeostasis Model Assessment of ß-cell function |
| IGT | Impaired Glucose Tolerance |
| IoM | U.S. Institute of Medicine of the National Academy of Sciences |
| LDL-cholesterol | Low Density Lipoprotein-cholesterol |
| LTI | Lower Threshold Intake |
| Metabolic syndrome | Cluster of cardiovascular risk factors including clinical measures such as waist circumferences, blood pressure, triglycerides, high-density lipoproteins, blood glucose, and insulin sensitivity. Usually three criteria are needed for the diagnosis of Metabolic Syndrome |
| MI | Myocardial Infarction |
| MUFA | Monounsaturated Fatty Acids |
| NSP | Non-Starch Polysaccharides |
| NNR | Nordic Nutrition Recommendations |
| PRI | Population Reference Intake |
| QUICKI | Quantitative Insulin-Sensitivity Check Index |
| RI | Reference Intake ranges for macronutrients |
| SCF | Scientific Committee on Food |
| SCFA | Short-Chain Fatty Acids |
| SD | Standard Deviation |
| SFA | Saturated Fatty Acids |
| SGLT 1 | Sodium Glucose Transporter 1 |
| STRIP | Special Turku Coronary Risk Factor Intervention Project |
| Sugars | Term conventionally used to describe mono- and disaccharides |
| Sugar | Term used to describe sucrose |
| TG | Triglycerides |

| | |
|---|---|
| UL | Tolerable Upper Intake Level |
| US | United States |
| VLDL | Very low Density Lipoproteins |
| WCFR | World Cancer Research Fund |
| WHO | World Health Organization |